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ACID BURNS OF THE STOMACH

REPORT OF TWO CASES*

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THE INGESTION OF CORROSIVE SUBSTANCES either suicidally or accidentally is not a rare occurrence. Nearly every physician has at one time or another been called upon to treat one of these cases; either in the acute phase or its sequelae. Cicatricial constriction of the esophagus caused by lye or other strong alkalis is a common occurrence. Anyone who has been associated with a charity service at any general hospital has had occasion to observe and treat such a condition. Although there are cases reported in which there is an associated esophageal stricture with lesions in the stomach, strictures of the stomach with an undamaged esophagus seem to be quite rare. An uninvolved esophagus with lesions demonstrable in the stomach have been found to occur more frequently following the ingestion of strong acids. A review of the literature reveals 140 cases reported in which pyloric stenosis has followed the ingestion of some corrosive, but only 12 of these were reported by American authors. In these reported cases there is a marked preponderance of stomach lesions where acid has been taken and of esophageal stenosis where the offending substance has been an alkali. This has been noted by all observers. Another constant feature of the recorded cases is the rapidity of onset of the stenosis. One case reported by Pop and Galdau[4] quoted by Schulenburg[11] had complete stricture in 19 days. It is the purpose of this report to review the literature briefly on all such cases, with special emphasis on those in which acid was ingested, and to report two cases in which the patients were observed and treated by us.

INCIDENCE

There is probably a large number of cases of ingestion of strong acids that are not reported in the literature because death ensues very shortly, and the late effects are not known. The earliest report of a gastric lesion from acid is that of Robert in 1828.[10] This patient swallowed sulphuric acid and died after nine weeks. Postmortem examination showed no pathologic lesion in the esophagus but there were ulcerated areas in the cardiac end of

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the stomach, and the pylorus was markedly contracted. In 1902 Quenu and Petit[9] collected 94 cases in which pyloric obstruction followed ingestion of corrosive substances. In 20 per cent of these cases there was some evidence of esophageal stricture. Early authors, including Orator,[8] reported about the same percentage of esophageal strictures associated with pyloric stenosis after ingestion of acid. However, since the earlier reports there have been 31 additional cases of pyloric stenosis caused by ingestion of acid. In only two, or 6.5 per cent, of these has there been evidence of esophageal stricture. The most common acids mentioned in these reports have been hydrochloric first and sulphuric second.

PATHOLOGY

Segments of the upper digestive tract show varying degrees of susceptibility to acids. In general, acids are strongly irritative and cause a coagulative necrosis of the surface epithelium and submucosal tissue. Blood vessels and lymphatics are destroyed and the tissues are left more vulnerable to continued exposure to the corrosive. This process may lead to immediate perforation if the acid is concentrated or if dilution and neutralization are delayed. Ordinary concentration of acid during its rapid course through the esophagus has but superficial scorching effect on the squamous epithelium lining the esophagus, but the delicate columnar epithelium of the stomach is much more susceptible to the acid and considerably more damage is done at this point. Dilution is a very important factor, and the ingestion of acid on an empty stomach will cause more extensive damage than will acid on a full stomach. However, the weaker solutions resulting from dilution may be tolerated and not vomited, so that the duration of exposure may be dangerously prolonged. Any portion of the stomach may be affected. In 1938 Testa[14] studied the gastric motility in dogs by adding barium to caustic soda and observed the progress of the mixture through the stomach by roentgen ray and fluoroscopic examinations. It was found that in the stomach the alkali flowed along the lesser curvature and a severe pre-antral spasm was produced. He demonstrated the severest trauma to be where the fluid had been held up by the pyloro-spasm. Grossly, the stomach is severely affected and in many cases the whole mucosal surface has a black, tarry appearance, or there may be areas of black altered blood, and severe inflammation of the adjacent part. Perforation may occur with the escape of gastric contents into the peritoneal cavity. Microscopically, destruction of the columnar epithelium, submucosa, and muscularis is seen. Early in the process there is necrosis of epithelium characterized by loss of the cell nuclei, with dilatation and thrombosis of regional blood vessels. There is little evidence of inflammatory reaction. This is soon followed by polymorphonuclear infiltration. These gradually disappear and a round cell infiltration, characteristic of chronic inflammation, takes place. Repair of surface epithelium may be expected but the rugae and gastric glands usually are destroyed. This thin layer of regenerated epithelium is likely to undergo ulceration. The entire stomach may be diffusely

affected, but the greater curvature, fundus and cardia will be relatively less scarred. In most cases, however, the only scarring is at the pyloric ring. Hour glass deformity of the stomach and other intermediate degrees of scarring are seen occasionally.

SIGNS AND SYMPTOMS

The onset of symptoms is immediate, with severe burning pain in the mouth, throat and stomach, spreading rapidly over the entire abdomen. Violent eructations and vomiting occurs, the vomitus being dark brown or blackish in color, mixed with altered blood and fragments of mucous membrane. There is intense thirst, with great difficulty in swallowing, and each attempt to drink is followed by renewed vomiting. Respiration is difficult and noisy; the voice is hoarse and speech may be impossible. Acute edema of the larynx may cause death from asphyxia. The mouth and lips are excoriated, the mucous membrane of which is sodden and discolored. The color depends upon the nature of the acid which has been swallowed. If the acid has been taken from a spoon or bottle the lips may escape injury. The patient usually retains consciousness, but the retching, vomiting, and destruction of tissue cause him to become rapidly weaker. Death may result from convulsions, suffocation, exhaustion or shock from perforation of the stomach. Death usually occurs within 12 to 24 hours from the local action, or partial recovery may take place and death occur after a few days from gastric inflammation. The patient may recover from the acute symptoms and die after two or three weeks from weakness and exhaustion, or he may live for years with stricture of the gastro-intestinal tract. In those cases where the reaction is not so severe, or the quantity of acid taken is not as great, recovery may follow and the patient be dismissed from the hospital in two or three weeks. In this type of case pyloric stenosis or other gastric lesions will frequently develop in four to six weeks. In such cases the patient will begin to vomit and lose weight rapidly. He will become quite dehydrated and a severe alkalosis will develop. The symptoms of a lesion in the stomach usually start with loss of appetite and a sense of epigastric fullness. As this obstruction progresses intractable vomiting ensues.

Roentgenologic examination is necessary to confirm the site and severity of the lesion, and to show the extent of secondary dilatation. Quite frequently the roentgen ray examination is suggestive of carcinoma of the stomach, and unless the history of ingestion of a corrosive substance is kept in mind the erroneous diagnosis of gastric neoplasm will be made.

TREATMENT

The use of a stomach tube carries some risk due to the softening of the tissue and the danger of perforation. Emetics are dangerous for the same reason. The first thing to be considered is neutralization of the acid, and for this purpose an alkali should be given. Calcined magnesia, lime water, or milk of magnesia may be used. Alkaline carbonates, which liberate carbon

dioxide, should not be used on account of gaseous distention of the stomach. Later, demulsive drinks such as milk, thin gruels, etc., should be given freely. When there is edema of the glottis, tracheotomy may be necessary. If circulatory shock, due to fluid loss and blood loss, has ensued, the patient should be given intravenous fluids and plasma. This should be continued until the feeling of thirst has passed, the mouth is moist, and urine is being secreted in sufficient quantities. After the onset of the sequelae from the ingestion of acids the treatment becomes entirely surgical. Four distinct methods of surgical attack have been used; they are as follows: (1) dilatation of the pylorus; (2) resection; (3) pyloroplasty; and (4) gastro-enterostomy. Very little can be said for the first method. This was tried many times in the earlier cases and fell into disrepute because of the uniformly poor results. Resection of the pylorus has been advised against by Schmieden[12] because of the difficulty encountered in suturing the damaged layers of the stomach wall. Pyloroplasty was practiced in Germany for quite some time but here again Schmieden's objection holds true.

FIG. 1.—(Case 1) Diagrammatic drawing showing extent of damage to stomach following ingestion of muriatic acid.

There has been only one recent case in which pyloroplasty has been attempted. This was reported by Arena[1] in 1936. His case was a two-year-old infant with stenosis of the pylorus following ingestion of muriatic acid. A Finney pyloroplasty was done with satisfactory results. Review of the literature revealed gastro-enterostomy to be the surgical treatment of choice for pyloric strictures secondary to ingestion of acids. Partial gastrectomy has been employed only four times in similar cases reported in the literature. The last such case being reported by Gray and Holmes,[6] in which partial gastrectomy was performed for pyloric stenosis following ingestion of sulphuric acid. The two cases reported here were treated by gastric resection.

REPORT OF CASES

Case 1.—E. L. T., 43-year-old white male, was admitted to the hospital October 2, 1947, because of severe diffuse abdominal pain of about 3 hours duration. Past history revealed that for the past 2 or 3 years this man had been a heavy drinker. He was a plumber by trade and on the day of admission he had installed a furnace in a dark basement. About noon, or approximately 5 hours prior to onset of symptoms, he came home very intoxicated, said that he had eaten his noon meal, and immediately went to bed. He slept until approximately 4 P.M. at which time he awoke and ate a rather heavy meal of beans and cornbread. One hour later he complained of severe pain in his abdomen which steadily increased in severity until admission to the hospital at

approximately 8 P.M. In looking through his plumbing kit two identical half pint whiskey bottles were found. One contained about 1 oz. of whiskey and the other one was about one-third full of muriatic acid.

The patient gave no history of vomiting from the time he went to bed at noon until onset of his acute pain at 5 P.M. After the onset of pain he took some soda and immediately vomited a large amount of dark red fluid which contained particles that looked like undigested food.

On physical examination he was seen to be a thin individual who seemed to be in excruciating pain. Blood pressure was 160/110; temperature 98.6°, pulse 88, respiration 20. Physical findings were limited to his abdomen. There was diffuse tenderness throughout his entire abdomen, with generalized "board-like" rigidity. Liver dullness was present; peristaltic sounds were not heard.

Roentgen ray examination revealed no free air in the peritoneal cavity either on the upright or lateral decubitus plates. Blood count: RBC 5,200,000, WBC 11,600, hemoglobin 15.9 Gm., polys 65 per cent, lymphs 44 per cent, stabs 3 per cent. Urinalysis showed a 1 plus albumin, specific gravity 1.022, sugar negative, acetone negative, bacteria 4 plus. Blood Kahn negative.

Clinical diagnosis of perforated peptic ulcer was made and the patient was taken immediately to the operating room and explored through a high right rectus incision. Upon opening the abdomen the stomach was found to be gangrenous from the pyloric ring to a point high on the fundus (Fig. 1). There was no gross evidence of perforation and no sign of volvulus of the stomach. There was much edema in the gastro-colic ligament. The duodenum and entire small intestine were normal. Nothing was felt along the course of the aorta to suggest an aortic aneurysm that could have occluded the celiac axis. The spleen was normal in size and color.

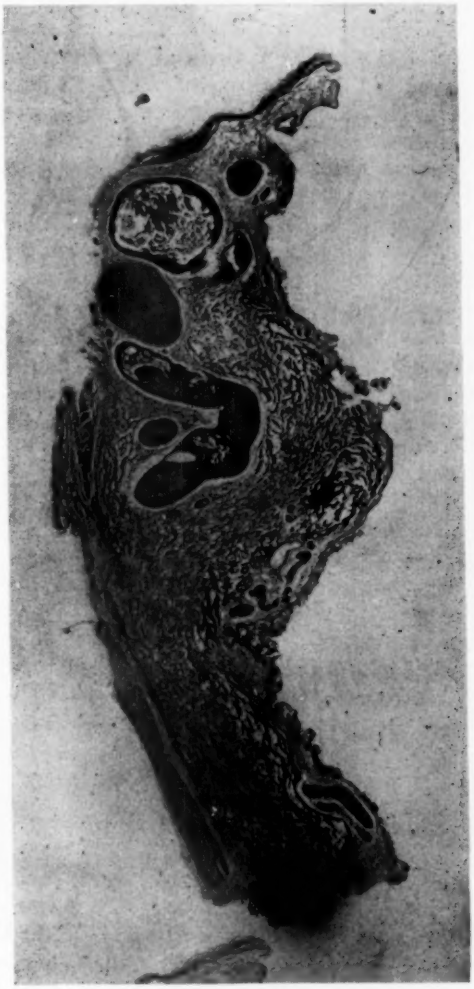

FIG. 2.—(Case 1) Photomicrograph of stomach showing necrosis of the epithelium with marked dilatation and thrombosis of the blood vessels. There is no inflammatory reaction.

An ante-colic, isoperistaltic, Polya gastric resection was done, removing approximately 85 per cent of the stomach. He made a good recovery and by the fifth day was taking liquid and soft food by mouth. The wound was dressed on the eighth day and the stitches removed. There was no sign of infection.

On the fourteenth day he had regained some of his strength, was tolerating an ulcer diet without difficulty, and he was discharged from the hospital with explicit directions as to diet and follow-up.

The pathologic report of the stomach is as follows:

Gross: The specimen consists of practically all of the stomach. The entire specimen is dark purplish red in color and the greater curvature is quite thick and hemorrhagic. The gastric mucosa is markedly discolored and has lost its normal velvety appearance.

Histopathology: Sections from each cut end of the stomach show extravasation of blood and degeneration, but very little inflammation. The blood vessels are filled with firm clotted blood. Sections from the thick greater curvature of the stomach also show tremendous extravasation of blood with necrosis, but with very little inflammatory reaction (Fig. 2). The absence of inflammatory reaction would indicate that the blood supply was completely obliterated. There is nothing suggesting old inflammation in the stomach or old fibrosis (Fig. 2). The blood vessels that are seen on the surface of the stomach are filled with firm clotted blood. *Diagnosis:* Extensive necrosis of the stomach, apparently due to a complete blocking of the blood supply.

The patient was readmitted to hospital on October 21, 1947, or 2 days after previous discharge. At this time he complained of difficulty in eating, inability to swallow, and loss of strength. He was somewhat dehydrated and emaciated; the abdominal wound was well healed, and there was no evidence of any weakness. RBC was 4,200,000, WBC 8,200, hemoglobin 13.1 Gm. (85 per cent) color index 1.01, specific gravity 1.020, and albumin was negative. Sugar was 3 plus (after intravenous glucose), WBC 6 to 8 per HPF. Blood was negative, epithelial cells 6 to 8 per HPF. There were no casts, bacteria or crystals. After admission to the hospital this patient was given intravenous fluids and blood plasma; his general condition improved quite rapidly and the day following admission he was started on an ulcer diet.

On October 27, 1947, a roentgenogram was made of his upper gastro-intestinal tract. Report is as follows: "Examination of the chest by fluoroscopy is negative. Examination of the upper G. I. tract was done with the patient supine and prone on account of his inability to stand. The esophagus presents a normal appearance. The stomach shows evidence of a subtotal gastrectomy with approximately 15 per cent of the stomach remaining. The stoma of the gastro-enterostomy appears to be functioning satisfactorily (Fig. 3). There is no definite ulceration or other pathologic change seen in the stomach at this time. A 4-hour film shows about 30 per cent gastric residue. The head of the meal had advanced to the cecum. *Impression:* Subtotal gastrectomy. The patient was dismissed from the hospital on October 28, 1947, very much improved and again was given explicit directions as to his diet and frequency of meals.

Nothing more was heard from the patient until November 10, 1947, or approximately 3 weeks later, when he was again admitted to the hospital. At this time he was very much emaciated and dehydrated. One week previously he had suffered some sort of abdominal accident with rigid abdomen and nausea; he was seen by a physician at this time who said that he appeared to be in shock. This episode lasted only a short time and he recovered rapidly. However, he continued to show signs of increasing inanition in spite of supplementary feedings by mouth. At time of admission his only complaints were weakness and a vague abdominal distress. Examination of abdomen at this time was entirely negative. RBC was 5,250,000, WBC 6,550, hemoglobin 15.9 101 per 100 color index 0.971, polys 72 per cent, lymphs 28 per cent, stabs 20 per cent. Urinalysis was unchanged from previous examination. Again he was started on intravenous plasma and given frequent feedings of high protein liquids. Large doses of vitamins B and C were given intravenously. He seemed to be improving and had gained a pound and a half in weight. Three days after admission he was started on a soft ulcer diet which he tolerated well. On November 19, 1947, or 9 days after last admission, he developed signs of pulmonary edema following intravenous glucose and died rather suddenly. Postmortem examination was done and reported as follows:

"*Gross:* The body is that of a markedly emaciated white man, appearing the stated age. Examination of the surface of the body shows no edema, no petechia or other unusual features. There is a healed surgical scar on the upper, right abdomen. The peritoneum is smooth and glistening, and the cavity contains no excess fluid. The pleurae are smooth. The trachea contains a large amount of thick, mucoid material which extends rather deeply into the substance of the lungs. There is only slight lobular pneumonia and no apparent edema. The pericardial cavity contains a slight excess of fluid. The heart is small and browner than usual. The coronary vessels and valves show no lesions. The aorta is smooth. The esophagus has a fusiform-like dilatation immediately behind the heart. The esophagus otherwise is not unusual. There is an anastomosis of the jejunum to the stump of the cardiac end of the stomach near the cardiac opening. This anastomosis is wide and there is no evidence of old or recent leakage into the peritoneal cavity. Immediately above the anastomosis and in the gastric mucosa

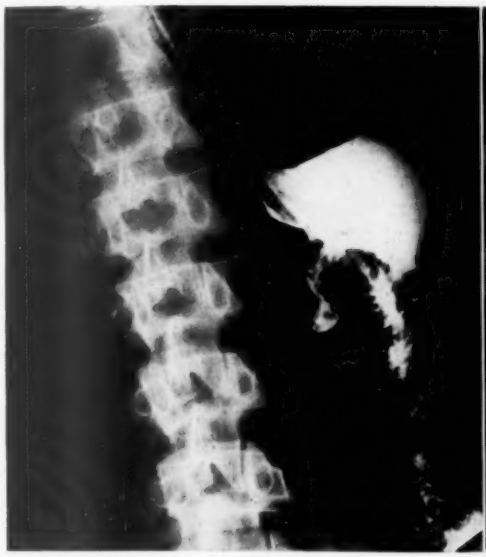

FIG. 3

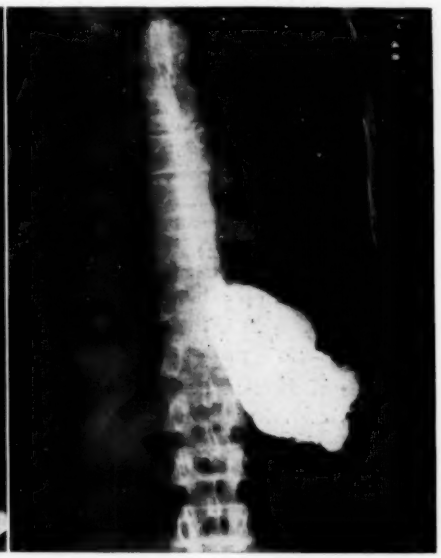

FIG. 4

FIG. 3.—(Case 1) Postoperative roentgen ray of stomach showing a well functioning gastro-enterostomy and no evidence of esophageal stricture.

FIG. 4.—(Case 2) Preoperative roentgen ray of stomach showing complete stenosis with no evidence of esophageal stricture.

there is a superficial ulcer, grossly triangular in shape measuring about 1 cm. on each side. The vestige of the stomach is attached to the spleen by old adhesions and there is a small walled off abscess at this point. The wall of the abscess is thick and fibrous. The small intestine and stomach contain cloudy fluid. The spleen is grossly normal. The adrenals are small and dark in color. The kidneys are approximately normal in size, but the surface of the cut sections show the pyramids to be quite dark in color. The capsules strip easily. There is no dilatation of the ureters or the pelvis. The pelvic organs are not unusual. The large arteries arising from the aorta were carefully studied. The celiac axis came off at a very sharp angle and was attached to and imbedded in the wall of the aorta for a distance of about 2 cm. The superior mesenteric artery came off at a right angle. There is no unusual atherosis in the aorta at this point. The low angle at which the celiac axis arises from the aorta suggests that pressure on this vessel

could have cut off the blood supply to the stomach. No other abnormalities in the blood vessels or other structures in this area could be demonstrated. The pancreas was grossly normal. The root of the mesentery is not unusual.

"*Histopathology:* Sections from the lungs show many of the alveoli to contain large, heart-failure cells. There is moderate inflammation in the mucosa of the medium and larger bronchi. Small islands of lobular pneumonia are apparent. In the heart the myocardial cells are small and many contain brownish pigment. The adrenal is not unusual. In the kidneys, many of the tubules are distended and contain considerable granular material. Some of the collecting tubules contain casts. The glomeruli and arterioles are approximately normal.

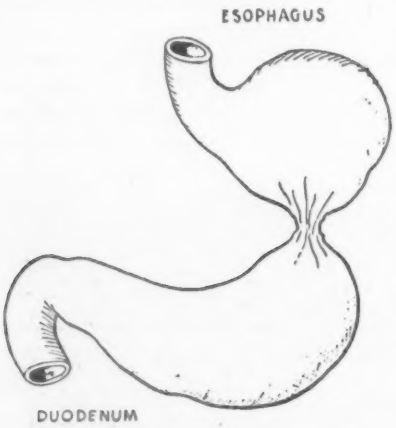

FIG. 5.—(Case 2) Diagrammatic drawing of the gross appearance of the stomach at operation.

"*Anatomical Diagnosis:* Emaciation, marked. Recent gastrectomy. Extensive collection of thick mucus in trachea and larger bronchi. Lobular pneumonia. Acute bronchitis. Fatty degeneration in myocardium. Gastro-jejunal ulcer. Old abscess between stomach and spleen, small. Parenchymatous degeneration in kidneys. The celiac axis arose from the aorta at a very flat angle, being attached to the aorta for about 2 cm."

Case 2.—E. M., 41-year-old white male, was admitted to the hospital on August 31, 1948, complaining of vomiting, weight loss, and severe weakness. Five weeks previously, while working at his gas station under the influence of alcohol, he reached for his whiskey but picked up a bottle of muratic acid by mistake. After taking two swallows he felt a severe burning in his mouth, throat and stomach. He immediately consumed a soft drink and vomited. He was taken to a hospital where a tube was passed into his stomach and its contents washed out. One hour had elapsed since ingestion of the acid. He was dismissed from the hospital 3 days later. Within 10 days he began to vomit most of his food except liquids. During the past week he has had epigastric distress, and has vomited everything taken by mouth. For the past five days he has been in a hospital where intravenous fluids were given and roentgen rays of the stomach were made.

Physical Examination: The temperature was 98°, pulse 80, respiration 18, and blood pressure 92/62. The patient was emaciated and too weak to walk. There were no ulcerations or scars of the mouth or pharynx. The heart sounds were barely audible. The lungs were clear. The abdomen was scaphoid and not tender.

Laboratory studies were as follows: RBC 5,600,000, hemoglobin 113 per cent, WBC 5900, polys 65 per cent, lymphs 35 per cent. The urine specimen was scant and insufficient for specific gravity determination. There was no albumin or sugar. Microscopic examination revealed 2 to 3 pus cells per H. P. F. The total proteins were 6.4 Gm. with A/G ratio of 1.5 to 1. The blood Kahn was negative. Gastric analysis revealed total acidity of 30° but no free HCL. Roentgen ray examination of the upper G. I. tract done on September 1, 1948, showed the esophagus to be free of any constriction. All the barium remained in the fundus of the stomach. The inferior border of the barium level had a rather scalloped appearance. The antrum and duodenal cap were not visualized. Impression: findings were suggestive of advanced carcinoma involving distal half of the stomach (Fig. 4).

On September 3, 1948, the third day after admission, the patient was operated upon. The stomach revealed a complete obstruction at the junction of the middle and upper

third. The lesion felt hard and irregular, and involved the mid-portion for about 10 to 12 cm. (Fig. 5). The greater curvature was adherent to the left lobe of the liver and spleen by dense adhesions. The distal portion of the stomach and duodenum were normal. There were some enlarged nodes at the greater curvature. The liver was smooth. It was felt that a high resection would be necessary to relieve the obstruction. Due to the patient's poor condition a jejunostomy was done and the abdomen closed. His nutrition improved with intravenous glucose, amigen, plasma and whole blood transfusions and jejunostomy feedings.

On October 1, 1948, one month after admission, when the maximum improvement seemed to have been obtained, he was again operated upon. A combined thoraco-abdominal incision was made and this gave excellent exposure. The spleen was removed. A sleeve resection of the mid-portion of the stomach was done which included the

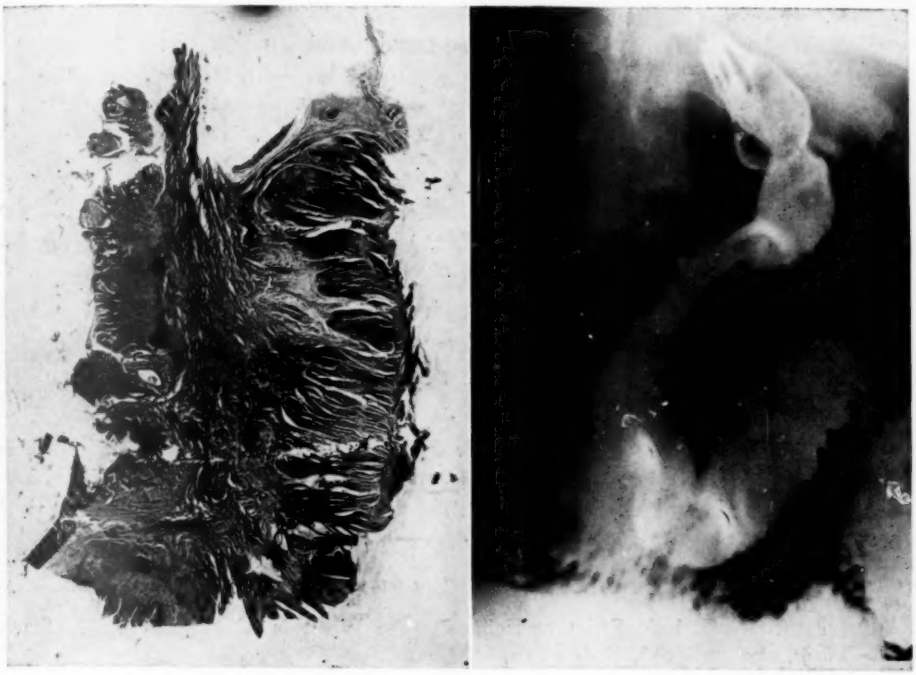

FIG. 6 FIG. 7

FIG. 6.—(Case 2) Photomicrograph of section through the region of the hour glass deformity showing a fairly good regenerated epithelium with marked scarring in the submucosa.

FIG. 7.—(Case 2) Postoperative roentgen ray of the stomach showing slight constriction at the point of anastomosis.

stricture and indurated area. The incision was closed and the patient left the operating room in good condition. He received 1000 cc. of glucose and 1000 cc. of blood during the procedure. Pathological report: complete stricture of the stomach due to scar tissue (Fig. 6).

On October 22, 1948, a roentgenogram of the stomach revealed a slight constriction at the site of anastomosis but the emptying time was normal (Fig. 7). He was dismissed from the hospital October 24, 1948. His progress was satisfactory except for inability to take average size feedings; however, he gained weight and worked every day. On February 3, 1949, he committed suicide by shooting himself.

SUMMARY

An attempt has been made to cover briefly the literature on the actions of strong acids upon the upper gastro-intestinal tract. Two clinical cases have been presented. Definite proof of the ingestion of acid in the first case is lacking but the history and subsequent pathologic findings are highly typical of changes shown following the ingestion of some corrosive substance. As this patient had in his plumbing kit both whiskey and muriatic acid in identical containers it is probable that he made an error in drinking the muriatic acid while working in a dark basement. The absence of local pharyngeal reaction cannot be satisfactorily explained. The thrombosis of all the vessels in the stomach as confirmed on microscopic examination is typical of local action due to some corrosive acid. We were unable to find in the literature a case brought to the operating room soon following the ingestion of acid. Reports of all other early cases were the result of postmortem examinations. In the second case there was a definite history of ingestion of muriatic acid with subsequent early treatment. The course of events in this case was typical of the recorded cases in which stenosis developed. However, this is the first case reported in which it was obligatory to do a gastric resection in order to relieve the obstruction. The outstanding feature of all recorded cases is the preponderance of gastric lesions where acid has been swallowed and of esophageal lesions where alkali has been taken. No rational explanation for this has been given. In the two cases reported there was no evidence of esophageal damage. In the literature 20 per cent of recorded cases with gastric lesions following the ingestion of acid also had esophageal stricture. A jejunostomy is often indicated. By this means fluid and electrolyte balance can be restored, and the state of nutrition improved for definitive surgical procedure.

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DISCUSSION.—DR. SAMUEL MCLANAHAN, Baltimore: I rise to compliment Doctor Strode on this interesting report on this always interesting lesion. The two cases he has reported are extremely unusual ones in this relatively rare condition; certainly very few have been operated upon as early as he operated upon the first case. Also, as he pointed out, relatively few resections have been done for this condition. Furthermore, he has given the pathologic changes which take place with the entrance of acid into the stomach. It has always been an interesting observation that the alkalies strike the esophagus and the acids have their effect upon the stomach.

Some years ago I reported such a case in a man who also was intoxicated, was somewhat depressed, and who secured some muriatic or dilute hydrochloric acid from a paint shop. These people seem to get their acid from paint shops and they all seem to be intoxicated! This man was brought to the hospital vomiting profusely and was treated conservatively for a time. It was our good fortune to secure a series of roentgen ray pictures at five days, at 12 days and at 23 days, which showed the progression of the lesion and, finally, the last pictures were interpreted—as Doctor Strode commented about his own case—as carcinoma of the stomach. Certainly it was a typical picture. We did not believe it was a carcinoma of the stomach, but that was the roentgen ray report.

(Slide) This man was operated upon and a gastro-enterostomy was performed. His postoperative course was satisfactory; he went along all right for 18 months, and he too came to a sad end. He was struck one night by a truck and sustained a fractured skull and other injuries which proved fatal. Fortunately he was brought to the same hospital and it was possible for us, 18 months after operation, to perform a postmortem examination, and this picture shows the artist's conception of the stomach.

The pyloric tumor was somewhat smaller at the time of death than it had been at the time of operation, but the pylorus was completely occluded. We filled the stomach with water, obliterating the gastro-enterostomy outlet, and no water whatsoever passed through the pylorus. This gave us an interesting view of the true pathologic entity, later. This man was treated by gastro-enterostomy and not by resection, because we felt it was a benign lesion.

DR. CHARLES A. VANCE, Lexington, Ky.: Of course I am interested in hearing this report because I saw the first case, and because the work was done by two young men who are my associates. I am pleased to hear them present a report like this. I saw the first operation; the stomach was just as black as your hat. I have been practicing for more than 40 years and I did not see how anything could be done with that stomach, but I stayed and watched and they took care of it in the right way. So I am much pleased to hear this report.

DR. MELVIN L. DEAN, Lexington, Ky. (closing): I wish to thank Doctor McLanahan for his discussion. We reviewed his paper very thoroughly when we were working on ours. I also want to thank Doctor Vance for his kind remarks.

RADICAL SURGERY IN THE TREATMENT OF CANCER*

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CLEMENT OF THE Alexandrian School, centuries ago, made a pertinent observation. He said "the beginning of truth is to wonder at things." Many thoughtful surgeons—and physicians—have wondered and still wonder whether the radical surgery of today in the treatment of cancer is justified. The purpose of this paper is to try to discuss the subject objectively and from the experience of one who has lived before and has been living in a period of the most active development of this radical surgery.

The horizon of the extent and radical nature of the surgery for neoplastic disease has been for the past 30 years, and continues to be, a widening one in all the anatomical fields. Operations are being done today by our leading surgeons, younger surgeons and residents that even 20 years ago were not attempted; and they are being done not only for cancer but to correct physiologic disturbances in various systems. The operative risk when many of these radical procedures were first attempted was high, but during the past ten or five years, has been reduced to the ten and five per cent level. At a recent conference in one of our municipal hospitals, the operative mortality for gastric resections for ulcer 20 years ago was reported as 25 per cent. During the past five years, the mortality has been 1.5 per cent. The operative risk has similarly been reduced in many other procedures; in lobectomy and pneumonectomy, in resections of the cardia and esophagus, in partial and total pancreatectomy, in colectomy, and in hemipelvectomy, to mention a few examples.

But in evaluating the validity and justification of radical surgery in the treatment of cancer in its anatomical distribution and organ involvement, certain factors must be weighed: (1) The threat of the disease, as to life, as to dysfunction, and as to disability and distress; (2) the operative risk; (3) the probability of cure; (4) the assurance of relief of symptoms even though palliative and temporary; (5) the ability of the patient to adapt himself or herself or to tolerate dysfunction and deformity, inevitable in some of the radical procedures; (6) the quality of the resident staff in the essential pre- and postoperative care of the patient; and (7) the experience, skill and integrity of the surgeon.

1. *Threat of the Disease.* Unless adequate therapy is given a patient with cancer, a fatal outcome is certain. In many such patients' dysfunctions, persistent and even intolerable pain and increasing disability are inevitable. Surgery is increasingly recognized as the most effective therapy in cancer. Cer-

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tainly this is true in the lesions of the abdomen, in the thoracic cavity, in the neurologic field, and in the extremities. Even in the field of the reproductive and genito-urinary systems, surgery by qualified general surgeons is coming to replace radiation therapy. But the surgery must be radical enough to leave no residue.

2. *Operative Risk.* Weighing against the threat of the disease is the threat of the risk of surgery. As has been stated, the operative risk is steadily decreasing in almost all of the radical procedures. To name a few of these we cite the following examples. When hemipelvectomy was first attempted the operative risk was in the neighborhood of 40 per cent. At a recent conference at the Memorial Hospital, Dr. John Morton reported eight consecutive procedures of this type without an operative mortality, and Dr. Coley and Dr. Pack reported 27 consecutive cases from the Memorial Hospital with a similar result—no operative mortality. In the first ten years of radical pancreatoduodenectomy at the Presbyterian Hospital in New York, the mortality was approximately 33 per cent in some 30 patients. During the past two years, Dr. Parsons and Dr. Lockwood[1] report 17 such consecutive procedures without an operative death. Dr. Cattell[2] in Boston has reported 40 radical operations for ampullary carcinoma with only one death.

Dr. Joe Meigs[3] of the Massachusetts General Hospital has supplied me with the following data on 46 cases where pelvic exenteration was carried out by Dr. Meigs and Dr. Langdon Parsons.

	Dead	Alive
Bladder retained	0	4
Rectum retained	2	7
Total exenteration	22	11
	24	22*

Died within one month	25%
Died in hospital	33%

PATHOLOGY

Carcinoma of cervix	36
Carcinoma of ovary	3
Carcinoma of rectum	3
Sarcoma of vagina	1
Carcinoma of Bartholin's gland	1
Carcinoma of cervix and carcinoma of rectum	1
Adenoacanthoma	1

LIVING OVER ONE YEAR

2 ..... 21 months	2 ..... 17 months
1 ..... 20 months	1 ..... 14 months
2 ..... 19 months	1 ..... 13 months
1 ..... 18 months	1 ..... 12 months

* Eleven living for more than one year.

Dr. Brunschwig[4] tells me that at the Memorial Hospital since September, 1947, there have been approximately 200 cases of radical panhysterectomy

with pelvic lymph node excision, with a surgical mortality of one patient (one-half of one per cent). These were unselected as to size of lesion, local extension, age of patient, weight of patient and nutritional status.

There have been 62 complete pelvic exenterations, and among these there are four patients who have lived more than two years without evidence of recurrent disease and three patients who have survived from one year to one year and ten months without evidence of recurrent disease. This latter group was of the extreme advanced and hopeless type.

The reasons for these great improvements in various fields are a better understanding and intelligent use of transfusion, fluid and electrolyte balance; improved anesthesia; care in minimizing tissus trauma, and of improved technics; in better exposure and better wound repair; intelligent use of the chemotherapeutic and antibiotic agents; and, perhaps more important than any factor, the far more experienced and intelligent pre- and postoperative care of the patients in preventing operative and postoperative complications by the adequately trained resident staffs in our best surgical clinics.

3. *The probability of cure and long term survival.* The salvage rate in the surgical treatment of cancer varies greatly, depending upon the type of tumor, the anatomic site, the limitation of spread, the radical nature of its removal, and the technical ability of the surgeon. Also, it depends upon his understanding of the natural history and behavior of cancer, and the importance not only of removing it and its spreading pathways *en masse,* but of treating the patient as an individual human being and not merely as a case to be removed from a lump of cancerous tissue. A surgeon trained in the pathology of cancer knows that the fungating type of carcinoma gives a better prognosis than the invasive, infiltrating variety, and remains localized for a longer period than the latter invasive type. And yet every cancer has individuality and is not altogether predictable.

Many examples of these factors determining the prognosis in the surgery for cancer could be given, but one lesion will be discussed. Carcinoma of the stomach is one of the most common and serious cancers encountered. The overall salvage rate is low. For many reasons, it is treacherous because of its insidious onset, its masking symptoms, its tendency to lymphatic spread, probably accelerated by the powerful contractions of its strong muscular coats, and the relative frequency of the invasive, infiltrative type of growth. The fungating type that mushrooms into the lumen and gives filling defects in radiologic examination and a palpable tumor on palpation, is the more easily diagnosed at an earlier stage. But even today the medical profession is entirely too pessimistic, and in too many favorable cases the patient is sent to the surgeon when the cancer has spread to the liver and extensively into the lymphatics. But to quote indirectly from a recent follow-up review of the gastric resections for carcinoma at the Columbia-Presbyterian Clinic by Drs. St. John and Harvel[5]: Of 256 patients with gastric resection for carcinoma of the stomach, 56 are living five years or more without evidence of recurrence, 32 are living between five and ten years, and 24 have survived ten years or

longer. One of these, whom the author resected, lived 21 years and died of cardiac disease. Of the 56 five year survivors, 15 showed lymph node involvement. All of these specimens had been carefully reviewed and re-examined by Dr. Arthur Purdy Stout, an authority on neoplastic disease.

At the Memorial Hospital, a study of the end results in the treatment of gastric cancer by Drs. Pack and McNeer[6] gives the following facts in patients studied from 1916 to 1946.

a. The resectability rate for gastric cancer has shown progressive improvement for each succeeding period: 1916-1930, 2.9 per cent; 1931-1936, 7.7 per cent; 1937-1941, 26.2 per cent; 1942-1946, 39.8 per cent.

b. Of 75 patients surviving gastrectomy performed five or more years ago, 26 or 34.7 per cent lived five years or more without recurrence.

c. The presence of perigastric lymph node metastasis of resected gastric cancers influences the end results of treatment. Those patients without nodal metastases had 42.8 per cent five-year survival without recurrence; those patients with proved nodal metastasis had 24.2 per cent five-year survival.

d. The operative mortality for subtotal gastrectomy for cancer has decreased with each succeeding surgical period. 1916-1930, 62.7 per cent; 1931-1936, 33.3 per cent; 1937-1941, 15.5 per cent; 1942-1946, 9.6 per cent. More than 80 patients in the period of 1937-1946 have had either total gastrectomy or transthoracic cardiectomy for gastric cancer.

e. Local serosal penetration resulting in fixation to adjacent organs whose removal is compatible with life may not be an unfavorable complication, perhaps because of the more radical operation that must necessarily be done. In this series of 16 patients with this complication who had these radical operations, eight were living and well at the end of five years, or 50 per cent. These specimens were examined by Drs. Stewart and Foote, authorities in neoplastic lesions.

4. *The assurance of relief of symptoms, even though palliative or temporary.* This more radical surgery has to be considered from both the curative and the palliative standpoints. Palliative surgery must not be justified in terms of months or years of survival, but rather by relief of intolerable symptoms, freedom from discomfort, and the ability to resume a relatively normal way of living. It is with this measure of well-being that the present radical surgery of pelvic evisceration for recurrent carcinoma of the cervix, following repeated bouts of radiation therapy, with the recurrence localized to the pelvis, must be judged.

These patients have constant pain, requiring morphine, vesico-vaginal or recto-vaginal fistula, or both, and are permanent invalids, often bed-ridden, and have been pronounced incurable. Pelvic eviscerectomy, including removal of the pelvic peritoneum and iliac lymphadenectomy, and requiring uretero-sigmoidostomy and a wet colostomy necessitating a special Rutzen bag, has to be carried out; and yet I have seen eight patients living one to two years after operation, all of them expressing satisfaction in living free from pain and carrying on their household duties. None of them gave evidence of recurrent

cancer. These procedures are being done in Boston and New York with a steadily decreasing operative risk.

Many esophagectomies and pneumonectomies, as well as pancreatic resections for cancer, must be considered palliative. But relief from intolerable symptoms even for a year or two is certainly everyone's right, if it can be given with relative safety.

5. *The ability of the patient to adapt himself or herself, or to tolerate the dysfunction or deformity inevitable in some of the radical procedures.* This is a problem involving the psychology of the patient as well as the ability of the surgeon and his associates to understand this psychology and to prepare the patient for the psychic trauma and to train the patient and the family in the control of his or her dysfunction. This is especially true in patients requiring colostomies, extensive amputations, laryngectomies, and in women requiring hysterectomy or radical mastectomy. A program for rehabilitating patients in these and other categories, physically and psychologically, has been organized at the Massachusetts General Hospital in Boston and at the Memorial Hospital in New York. Interesting studies in the care of these patients, in the training of the residents, nurses and social service workers who deal with these patients, and in research in the psychologic reactions of the patients, are developing, and valuable contributions will certainly be made in these fields. A very effective rehabilitation project, which the members call the "Amputees' Alliance" under the supervision of Dr. Bradley Coley at the Memorial Hospital, has been functioning for two years in convincing patients requiring amputation, even hemipelvectomies, that an amputee can be a useful and happy citizen. In the past, too many surgeons have paid too little attention to the psychologic and physical rehabilitation of cancer victims.

6. *The quality* (I emphasize the term "quality") *of the resident staff in the essential pre- and postoperative care of patients undergoing radical surgery for cancer.* This is a factor the importance of which cannot be overemphasized. Carefully selected residents, with adequate training for periods of four to five years in general surgery, with an intelligent understanding of the adjuvants previously mentioned that have done so much to lower the hazard of surgery, are essential in any hospital attempting to do present day radical surgery.

The "alarm reaction with the break-up" of the patient immediately after extensive surgery and the "put together" of the postoperative period, so graphically described by Francis Moore, involve complicated processes of fluid and electrolyte imbalance and abnormal adrenal response which have only in the last two or three years been studied. The syndrome of metabolic alkalosis with potassium depletion has only recently been described, but is a most important and serious complication in some of the radical and extensive procedures. It is now recognizable by serum potassium determinations and electrocardiographic studies and readily prevented or controlled by the administration of potassium. It is the well-trained, intelligent resident, aware of the recent advances in the prevention and treatment of complications, that is the indispensable assistant

of the surgeon doing this type of surgery. Such a resident is often more competent than a so-called specialist who has not had residency training in general surgery.

7. *The experience, skill and integrity of the surgeon.* These characteristics of the surgeon doing this type of surgery determine his essential quality. His experience determines his qualification for deciding the extent and radical nature of the surgery to be done and the care of the patient before and after operation. His experience determines his judgment in deciding on whom and when to advise it, and how to deal with patients in advising them, for they differ individually in their reaction to such advice and to the way it is given. This applies to the family of the patient as well. His skill determines to a large extent his ability to carry out difficult and hazardous surgery; but this does not mean the speed with which he operates so much as the care of his tissues and the minimum trauma he inflicts in the doing of the surgery, and the repair he accomplishes. However, it does not mean puttering and wasting time.

His integrity is more difficult to define, but we all know what it is—we know when a surgeon has it and when he has it not. It implies humaneness and kindly understanding in dealing with the cancer victim, his family and his problems, his ability to restore confidence and hope, and his desire to use infinite pains in caring for the patient, and not considering him as a case to be removed from a cancer. To have integrity, he must know his limitations and be willing to advise the patient to seek the services of a surgeon capable of handling the problem.

Another most important attribute of the surgeon is his interest and enthusiasm in the training and supervision of his residents. His example, if good and characterized by an unselfish devotion to his patients, with constant desire to improve his surgery and to do sound research, is the greatest stimulus to the younger men working with him. Witness the amazing influence of Halsted on his residents and their desire to hand it on to following generations of residents.

One of the hazards of radical surgery at the present time, especially in some of the newer procedures, is the tendency on the part of certain specialists without experience in the field of general surgery to visit a clinic where some of the extensive operations are being done, to see one or two such procedures, and then to return to their own clinics to begin this work. The results will unquestionably be calamitous but will not be published. This type of surgery cannot be learned from a surgical amphitheatre or by television, and should not be attempted by those who have not had adequate training and apprenticeship in general surgery, in the preoperative, the operative and the postoperative care of the patients subjected to such surgery. This discussion is closed with this warning.

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DISCUSSION.—DR. HARVEY B. STONE, Baltimore: May I say to begin with that I am delighted that Doctor Whipple has honored the Southern Surgical Association with his presence and with his excellent talk; and further, that he has always been, through the many years I have known him, one of my outstanding idols in surgery and one of the men on whom I have endeavored to model myself.

I should like to say further that some of the comments Doctor Whipple made, I think, were directed more toward the consideration merely of extensiveness in the field of surgical procedures, and not limited to the surgery of cancer. For instance, I think one has to adopt a different criterion of judgment as to the rationale of the extensiveness of an operation devised for the purpose of the cure of cancer, from that devised for the correction of an anomaly, such as Doctor Blalock has done in cardiac anomalies. And I hope to make clear the basis for that differentiation in a few comments later on.

Let me say again, if all the proponents and practitioners of the idea of extensive surgery in the treatment of cancer (to which I would like to confine my remarks) were as gifted with the wisdom, the balanced judgment, and the character that Doctor Whipple has displayed, not only in his talk but in his life, much of what I have to say would be purposeless. But under the existing circumstances I do wish to make a few comments. In the first place, the trend, now beginning to be widespread, to extend the limits of destructive surgical attack on carcinoma, I think, needs critical appraisal. I may say here that Doctor Whipple has just subjected it to such appraisal. I would like to continue that consideration briefly a little further. One must admire the courage and the technical craftsmanship of those who are engaged in this extension of the field of surgery. But also I think one may question whether some of the impetus does not derive more from pride of craftsmanship and response to challenge, than it does from a reasoned decision on the merits of the individual case concerned. And while these efforts have definitely widened the scope of possible surgical ablation they have not in any degree introduced a new principle into the treatment of cancer. The surgical principle remains the same, and may be briefly stated as follows: the removal of the disease as extensively as possible with due regard to the survival and the subsequent state of existence of the patient. It is the latter part of this surgical principle, it seems to me, that is somewhat overlooked in the present tendency. The salvage of these cases over long periods of time is admittedly small. The immediate loss is quite large and, of the survivors, many are mutilated and functionally crippled to such a degree that certainly their existence cannot be regarded as satisfactory. The cancer, and not the patient, is sometimes the object of the treatment, and I submit that that relationship should be reversed. Almost in the words of Doctor Whipple, it is not a question of removing the patient from the cancer; it is a question of treating the patient in the best possible way in view of his cancer.

The economists have a phrase—"the point of diminishing returns"—and by that they mean that there comes a time in the economic processes where the cost of the procedure exceeds the benefits obtained from it. And again I submit that in some of these extensive procedures for the treatment of cancer the cost to the patient exceeds the benefit that he may possibly expect to obtain. The trend in this direction of widespread excisions may cause great damage, I think, to many individual people and to the good name of surgery, unless it is subjected to careful, critical restraint. Please note that I make no objection whatsoever to the extension, the radicalness, the widespreadness, of the procedure *per se*. I only ask that it be employed with due and proper concern for appropriateness in any given case. And to apply such procedures—as is being done,

I am quite sure—in cases where there is obviously the barest possible chance of benefit to the patient, seems to me to be certainly an almost indefensible proposition.

I should like to end with one comment. I have heard, and I have read in some of the publications, from the proponents of this attitude toward the field of cancer surgery, a defense which is based upon something of the sort which follows—without recalling individual citation: Doctor Welsh and Dr. Nicholas Senn, and other eminent figures are quoted as showing that in the past they have adversely criticised proposals of their own day, and the passage of time showed that these criticisms were inappropriate and mistaken. It is no defense of any current field of action to say that past criticisms in other fields of activity have proved to be false. The case under discussion must stand on its own merit, and again I submit that what is needed right now, in the face of this trend, is balanced, courageous, sound criticism.

Dr. Joe V. Meigs, Boston: It is rather difficult for me to say much of anything about this problem. All I know is, and all I can say is, that looking at the treatment of cancer of the cervix—a disease in which I happen to be interested—from a perfectly cold-blooded point of view, one cannot help but realize that the present type of treatment has very definite and distinct limitations. I started the advocacy of the return to radical operation for cancer of the cervix by reading a paper concerning such surgery in 1945, and sometimes I am very sorry for having done so. I feel that perhaps radium and roentgen ray therapy are better than surgery; on the other hand, we can report at the present time from our hospital about 200 patients who were subjected to what we considered a truly radical operation without mortality. This type of surgery must be radical. We have had no deaths and I feel that our record can stand comparison with roentgen ray and radium treatment. It is true that we have had ureteral fistulas, but so have those advocating radiation. I cannot say which is better or which is worse. I don't know. Dr. George Waterman of Providence, who has obtained as good results with radiation as anyone in this country, reports 70 per cent of five year cures with roentgen ray and radium in the Stage I cases. At the end of five years our figures by surgical approach are 80.4 per cent salvage. These figures will vary, I am sure. In our surgical group there were 11 patients who had positive lymph nodes and three are living for five years. I consider that something of an advance.

The difference between radium and surgery in the Stage I and Stage II patients is not the point. It is in the more advanced cases—the Stage III's and the Stage IV's. Stage III means involvement of the broad ligament out to the pelvic wall; and Stage IV means involvement of the bladder and the rectum, or even more advanced disease—cases for which practically nothing can be accomplished by treatment with x-ray and radium. Doctor Waterman's results in Stage III are 7 per cent cured for five years. Our results at the Massachusetts General Hospital and the Pondville Hospital (Massachusetts Department of Public Health) in nearly 2000 patients are 12 per cent five year salvage. Doctor Waterman's Stage IV's show zero per cent of cures at five years; in our hospitals, 4 per cent. It is very difficult to have to face Stage III and Stage IV patients in the office and tell them that all one has to offer is roentgen ray and radium, and that the results are about 10 per cent in one group and zero per cent in the other group. It seems to me that some sort of a better method must be devised for handling these particular groups.

When I started to do radical surgery for cancer of the cervix it was my intention to advance from Stage I to II, to III, to IV, but Doctor Brunschwig commences his surgical attack on the Stage IV's, III's, II's and I's; he has reversed the direction. He has taught us a lot. Our group studying this problem of ultra-radical surgery has associated with it all those interested in electrolyte balance, the pediatric service, the gynecologic service, the surgical and medical services, and those interested in the liver, in transfusions, and in endocrine disease. The psychiatric department is in full consultation with us. Our results are not good. We have a 25 per cent mortality, including Dr. Langdon

Parson's cases, and he has a larger number than I have; his group and mine constitute a fairly good example of what is going on. Twenty-five per cent of the patients we operated on have died. At the end of one year we have 22 of 46 patients living, 11 of them living more than a year. I can only tell you that so far as I can see, those patients have, in the main, been rehabilitated. To be sure they may be uncomfortable, some with wet colostomies, some draining all urine by rectum, and some with skin ureterostomies, most of them without any vulva, without any vagina, without any bladder or rectum. On the other hand, these patients were miserable; roentgen ray treatment could do no more and radium could do no more.

What are you going to do for a patient who is so uncomfortable that she is a morphine addict? If you could see the picture of Alex Brunschwig's "First Year Club," the first six patients he operated upon over a year ago, you would see those patients who have gained from 20 to 50 pounds each and see a group of women smiling and laughing in the photograph (maybe they were laughing with Alex, who knows?), but at least they are an entirely different group from the bedridden women upon whom he was operating. I feel with Doctor Stone, for whom I have the greatest admiration, and Doctor Whipple, for whom my admiration knows no end—I am glad they both realize that this is not an operation to be done universally. This is really an experimental procedure and I hope it will be confined to a few of the large clinics or university clinics that are really interested in this problem.

Dr. Bradley L. Coley, New York: Doctor Meigs has stressed the place which radical surgery occupies in the treatment of advanced cancer of the cervix; I shall confine myself to a discussion of only one phase of the general subject.

In deciding for or against a radical operation for cancer, and especially such mutilating procedures as hemipelvectomy and interscapulothoracic disarticulation, there are many factors to be taken into consideration. First, what is the patient's course going to be without operation; that is, the extent of his suffering and duration of life? What is the probable life expectancy with operation, and does the period of time justify the procedure? Will neurosurgical relief of pain be preferable to an operation which envisages removal of the tumor, e. g., chordotomy, prefrontal lobotomy, or topectomy? How comfortable and happy—and I emphasize the word *happy*—can the patient be made if the operation is performed? Is there an acceptable alternative method, such as roentgen ray therapy? All these questions must be answered by the man who has to make the decision.

I had always been strongly opposed to hemipelvectomy, but shortly after my return from service I decided that we ought to give it a trial on the Bone Tumor Service of Memorial Hospital, and in the past 37 months we have performed nine of these operations. All but one was a primary bone sarcoma involving the pelvic bones. The exception was a high femur tumor with a pathologic fracture and was totally beyond a hip disarticulation. There were no operative deaths in this group of nine cases, but three of the patients have since died and two are dying. Of the four living patients, one has survived three years and one month, one two years, one a year, and one nearly five months. Two of these patients are working, one operating a bar and grill, and the other performing her household duties satisfactorily. Incidentally, the latter is the earliest case, who was operated upon 37 months ago; while she was regarded as one of the most unfavorable cases to start with, she has certainly proved a most gratifying one.

I had always assumed that after hemipelvectomy it would be impossible to fit the patient with a prosthesis that would be even remotely satisfactory, yet two of our patients get about quite well with a special type of artificial limb; one has worn his for nearly a year.

We hope that with better judgment in the selection of cases for operation—based not merely upon the extent of the disease but on its histologic characteristics as well—we will establish a more practicable basis that will aid us in advising these unfortunate patients. Further studies of larger series of cases, perhaps the series of several surgeons

combined, should give us sufficient experience to help us in determining the place that this procedure should occupy as a means of handling carefully selected cases of cancer of the hip and pelvic bones. Doctor Meigs has given you a graphic picture of the misery of his patients with extensive disease in the pelvis which has prompted him to employ radical surgery. Anyone who has had the unpleasant experience of watching a patient with a slowly-growing chondrosarcoma of the ilium for whom radiation is so ineffective, will be prompted to try something which offers a prospect of relief of pain and at least a remote possibility of long-standing survival. The only alternative at present involves a neurosurgical procedure, such as chordotomy or topectomy.

DR. HUGH A. GAMBLE, Greenville, Miss.: I feel somewhat at a loss coming up before these authorities on this subject, but the question of hemipelvectomy has been brought up. I have yet to see a malignancy of the lower extremity in which the glands of the pelvis were not involved. Personally, I have performed five hemipelvectomies and in every case the pelvic glands were involved; three are dead, two are living. But I feel that those two did not have any chance at all without that operation. Hemipelvectomy in that type of lesion is, you might say, mandatory, in my opinion.

DR. FRANK H. LAHEY, Boston: Having recently written an editorial advocating total gastrectomy in place of subtotal gastrectomy for cancer of the stomach, it seems to me that when radical surgery is being discussed, I should rise and defend my position regarding this part of the subject. Irrespective of this advanced surgical method of dealing with extensive malignant disease, we can all say today that subtotal gastrectomy for cancer is an inadequate procedure in terms of the type of surgery that we apply to cancer elsewhere. If we were to do the same type of operation today for cancer of the rectum or colon that we do for cancer of the stomach, we would be criticized vigorously and, I think, very properly. In this editorial I have said that if you will recall the node-bearing area of the stomach, with these nodes extending from the pylorus along the lesser curvature up to the point where the esophagus goes through the diaphragm, and along the greater curvature, particularly the group between the greater curvature and the hilum of the spleen, and their distribution in the great omentum, and at the same time if you place the line of transection in the stomach at the usual site chosen for subtotal gastrectomy for carcinoma of the stomach, you would probably be willing to admit that in terms of radicalness this is an inadequate operation.

I have hesitated to take this position until I could answer three very appropriate questions in the affirmative. They are: Is the mortality low enough (we have now done 133 total gastrectomies) so that it is reasonably comparable with that of subtotal gastrectomy? It is now practically the same. Next, have there been enough five-year survivals in these very advanced cancers of the stomach to which this operation has been applied, to justify its application in earlier cases of carcinoma of the stomach? We are not able to answer this question yet in terms of five-year survival, but in terms of three-year survival, recognizing that every case in which to date total gastrectomy has been done is a presumed hopeless case, it can be answered in the affirmative, as the three-year survival is 21 per cent. The third important point which one could properly make in opposition to this position is, can you feed these patients satisfactorily? One cannot answer that completely in the affirmative because there will be occasional patients after total gastrectomy in whom there will be trouble, but their number is diminishing progressively as we learn to do the operation better. If they do have trouble with regurgitation, as a few of them do, that diminishes progressively with the number of years the patient survives after operation.

While I would be the last to say that every surgeon should substitute total gastrectomy for subtotal gastrectomy, I do believe that we must become more aggressive than we have been with carcinoma of the stomach. When we realize that it has now been demonstrated so conclusively that submucosal spread of gastric carcinoma to the duodenum

from the pyloric region is definite, that adds another need to the aggressive approach to this problem, that is, the greatest possible removal of the duodenum together with total gastrectomy. By means of radical removal of the duodenum and total gastrectomy, we can remove all the gastrohepatic omentum, we can wipe down the nodes from beside the esophagus, we can take out the group of lymph nodes in the omentum, and we can remove the nodes between the greater curvature and the splenic region by the addition of splenectomy. I believe the five-year survival rate is now so low, and we have made so little progress over the years in the treatment of cancer of the stomach, that certainly in the hands of those equipped by experience and by operating conditions to undertake this procedure, we have reached the place where we should attempt to improve them by total gastrectomy. A group of cases can then be acquired which will let us know whether or not we cannot widen the radicalness of the surgical approach to this lesion and thus increase the five-year survivals.

It has been very interesting to me to listen to Doctor Whipple and to Doctor Stone. This discussion, while dealing with the problem of the very radical surgical approach to the management of advanced malignant disease, has interested me particularly as to his philosophic side. It is such a delicate subject, and one so incapable of being standardized in terms of when to operate and when not to, that there probably does not exist in surgery a comparable field in which, were we the victims, we would be more concerned with and interested in the character and the personality of the surgeon. It has been said repeatedly that the very foundation of surgery is surgical judgment, and perhaps in this field it has the greatest opportunity to demonstrate itself.

Dr. Evarts A. Graham, St. Louis: In answer to Doctor Blalock's request for some remarks from me, it is hard to know what to say. I am, however, old enough to be interested in seeing changes in trends of surgery. Those of my age or older will recall that along about the turn of the century it was customary to be extremely radical in the surgical treatment of cancer; in fact, hardly any procedure seemed too radical to perform in those early days. The mortality was terrific when one became too radical, and there was great question as to whether or not the results obtained were justifiable. Of course there were many things connected with operative mortality at that time which were not known. Practically nothing was known about the treatment of shock, for one thing. Nothing was known about what might be loosely called chemical tests for operability. A new generation of surgeons has come up since those days and this new generation has the courage, and also the additional knowledge, to undertake what their fathers, and in some instances their grandfathers, found was not worth while.

The real question, it seems to me, cannot be answered today; we shall have to wait several years before we can really know how many of the patients who undergo this very radical surgery today are alive five and ten years later. I think that will be a major test—not the only major test of course. There is also, as has been stated by Doctor Whipple and all the discussors, the question of whether or not life is made more comfortable for these people. I confess that I am in the middle of the road in my attitude: I don't know what the answer may be and I would not venture to consider myself a good enough prophet to guess. We do have many advantages today over what our fathers had at the turn of the century when we were doing radical surgery for cancer. We can certainly reduce the operative mortality, but whether or not it will be worth while only time can tell.

It is interesting to note how the pendulum swings again, as it does in so many of our attitudes, not only about medicine but about everything else. I recall vividly, and some of the men here will recall even more vividly, the important influence Will Mayo and Charlie Mayo—I think Will particularly—had in stopping the very radical surgery which was the tendency in their early days. They laid down certain criteria which made extensive operations inadvisable. I recall on many occasions visiting the Mayo Clinic and watching the two Mayo brothers operate, hearing comments from those present about the fact

that it was fine to see the Mayo brothers, with their wide experience, realize that nothing radical could be done in many cases after making an exploration. Now the pendulum seems to be swinging back to the period before their time. What will be the result?

Doctor Blalock was kind enough to mention the doctor, who is now somewhat famous, who had his whole lung removed 17 years ago next April, who is still living. Incidentally, he had two glands involved with the cancer. The experience of many others throughout the world who have tackled this difficult problem in surgery, I think, is sufficient to show that it is well worth while to carry out radical operations on people who have primary cancer of the lung if it can be done with a reasonable degree of safety. The anatomic conditions present, however, in advanced cases of cancer of the lung are such that one cannot make a proper dissection of the large vessels, because the cancer so frequently surrounds and invades the large vessels at the hilum of the lung that it is impossible to dissect out the vessels and therefore to be able to remove the lung.

I wish to thank you for your kindness in allowing me to be here and to participate in this discussion, Doctor Blalock, and I wish I had something more important to say about the subject which Doctor Whipple has so ably presented in his paper. In closing, however, I should like to add that the triumphal tour of Marion Sims in Europe many years ago, which was mentioned today, was duplicated by your president in 1947, our own Alfred Blalock. I know, because I was there and witnessed the homage paid to him by surgeons from all over the world.

DR. ALLEN O. WHIPPLE, New York (closing): I simply rise to thank those who have discussed this paper. I appreciate the discussion and I want to thank them for it. Thank you, Doctor Stone.

SARCOMA OF THE STOMACH*

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SARCOMA OF THE STOMACH is not a common gastric neoplasm, but such tumors do occur often enough that the physician and surgeon should be aware of their frequency and of their distinctive character so that they may be considered in the differential diagnosis of tumors arising in the stomach, and in particular these tumors should be recognized at the operating table, by their gross appearance or by an immediate frozen section. While it is true that the majority of sarcomas of the stomach are not diagnosed before operation, it is of great importance to consider sarcoma in the differential diagnosis of gastric tumors because sarcomas may arise in a somewhat earlier age group, not infrequently present themselves as palpable tumors in the epigastrium and often are massive neoplasms involving a considerable part of the stomach. The presence of a large palpable abdominal mass may make the examiner consider these tumors to be inoperable carcinomas because of the extensive involvement of the stomach and because of their size. If certain characteristic features of these tumors are well recognized, however, surgical treatment may have much to offer in the way of relief of symptoms and even possible cure, since the prognosis is considerably better in sarcoma than in carcinoma. In this paper a clinical analysis is presented of 41 patients with sarcoma of the stomach seen in the Lahey Clinic between the years 1929 and 1949. During this time there were 1171 operations for malignant tumors of the stomach, making the ratio of sarcoma to carcinoma 1 to 28 or an incidence of 3.5 per cent. In this presentation we shall discuss also the pathologic features as well as the type of treatment employed and the results of follow-up studies in this group of cases.

The incidence of sarcoma of the stomach is given at variant figures by different writers on this subject.[3, 4, 11, 12] Ewing estimated that sarcoma comprises about 1 per cent of all gastric neoplasms. Balfour and McCann found the average ratio of one case of sarcoma to 111 of carcinoma, and in a total group of 4159 malignant lesions of the stomach there were 47 sarcomas. Warren and Lulenski found 28 cases of solitary lymphoid tumors among 3132 malignant tumors of the gastro-intestinal tract, an incidence of 0.9 per cent of sarcomas of lymphoid origin. However, in this group there were 14 primary lymphoid tumors among 569 malignant lesions of the stomach, an incidence of 2.5 per cent. In 1914, Forni made a complete and detailed analysis, and col-

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lected 200 cases of gastric sarcomas previously reported in the literature. D'Aunoy and Zoeller further reviewed the literature and found 335 cases of sarcoma recorded through 1929. Yarnis and Colp found 8 cases of lymphosarcoma in a series of 250 neoplasms of the stomach over a six-year period, an incidence of 3.2 per cent. Thus, while there is some slight difference in the incidence of sarcoma of the stomach as reported, it has varied relatively little. A survey of the literature conveys the impression that sarcoma of the stomach occurs fairly constantly and accounts for about 4 per cent of malignant gastric neoplasms and must, therefore, be considered in any differential diagnosis of tumors arising in the stomach.

PATHOLOGY

Sarcomas of the stomach may arise theoretically from any mesenchymal tissue component of the organ. Malignant tumors arising from fibrous tissue, fat and blood vessels are so rare that they are merely curiosities. For practical purposes, there are only two types of gastric sarcoma, those arising from

TABLE I.—*Classification and Incidence of Gastric Sarcoma (41 Cases).*

Leiomyosarcomas		9
Lymphoid tumors		32
Hodgkin's disease	10	
Reticulum cell sarcoma	6	
Lymphosarcoma	12	
Malignant lymphoma	4	
Total		41

smooth muscle and those arising from lymphoid tissue. The malignant lymphoid tumors may be further subdivided according to the criteria laid down by Warren and Lulenski. A classification of the tumors studied in this report is shown in Table I.

The malignant tumors of smooth muscle origin (Figs. 1, 2 and 3), the leiomyosarcomas, were located in various portions of the stomach but were more common in the pyloric third. There was no apparent predilection for greater over lesser curvature, or for anterior wall over posterior wall. The size varied from 2.5 cm. to 14 cm. in greatest diameter. The tumors, although sometimes lobulated, were in most instances well circumscribed. Usually they grew predominantly into the gastric lumen, but intramural or subserosal growth also occurred. The compressed and thinned overlying gastric mucosa in all but two instances showed one or more central ulcerations which were often quite deep, giving a punched-out appearance. Necrosis, either focally or involving the entire tumor, was common and resulted in softening the usual firm consistency. In all instances the tumor was thought to be completely excised locally; metastasis to lymph nodes was not found in any case.

Microscopically, the *leiomyosarcomas* arose from the gastric muscularis and were composed of spindle-shaped, relatively well-differentiated, smooth

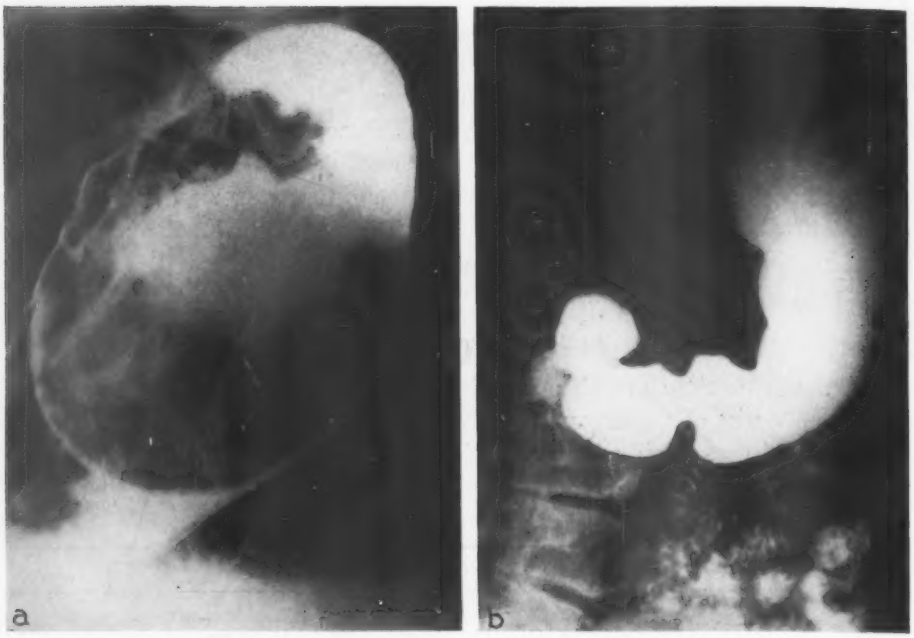

FIG. 1.—Leiomyosarcoma. (A) Intraluminal mass filling upper media and cardia; pedicle in upper cardia; transgastric excision performed. (B) Postoperative roentgenogram showing normal appearance.

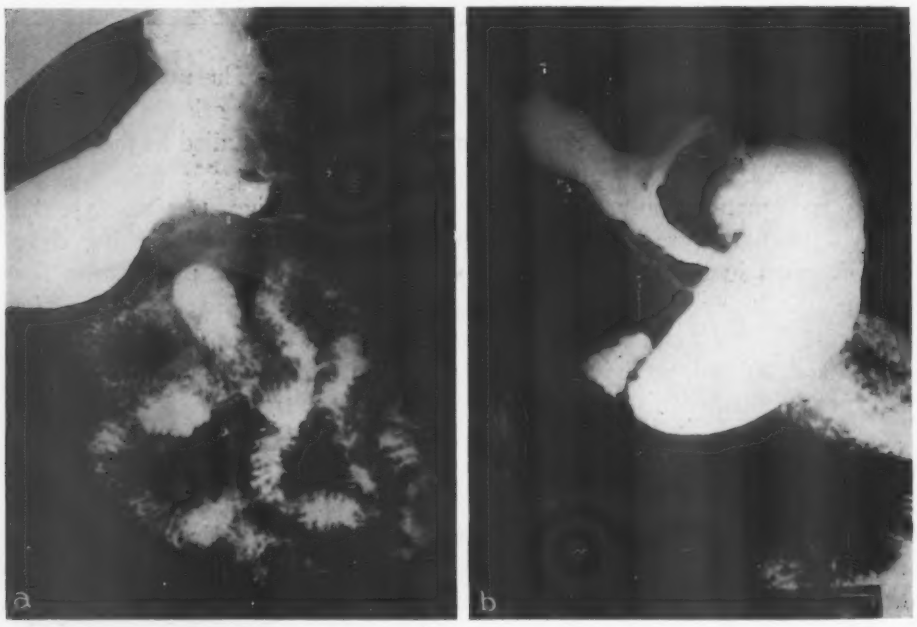

FIG. 2.—Leiomyosarcoma; the patient is well two years after operation.

muscle cells (Fig. 4). There was moderate pleomorphism of cells and nuclei. While scattered mitoses were present in each case, they were never numerous. The general impression was that of rather slow growth and low-grade malignancy. These tumors differed in microscopic appearance from benign leiomyomas, which they may grossly resemble, by showing invasion of normal

FIG. 3

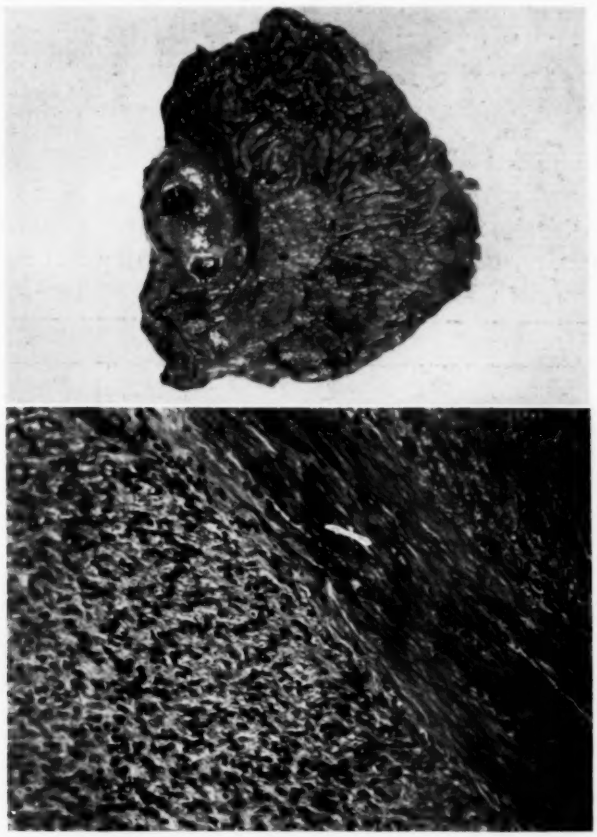

FIG. 4

FIG. 3.—Leiomyosarcoma. The tumor is growing into lumen and shows multiple typical mucosal ulcerations.

FIG. 4.—Microscopic appearance of leiomyosarcoma. The tumor is growing on either side of normal strand of muscle (eosin-methylene blue stain, x 250).

tissue and evidence of more active growth. No invasion of blood vessels or lymphatic vessels was found.

The *lymphoid tumors* (Figs. 5-8), of which there were 32, like the leiomyosarcomas were located in various portions of the stomach and showed no predilection for either wall or either curvature. The majority of the tumors

FIG. 5

FIG. 6

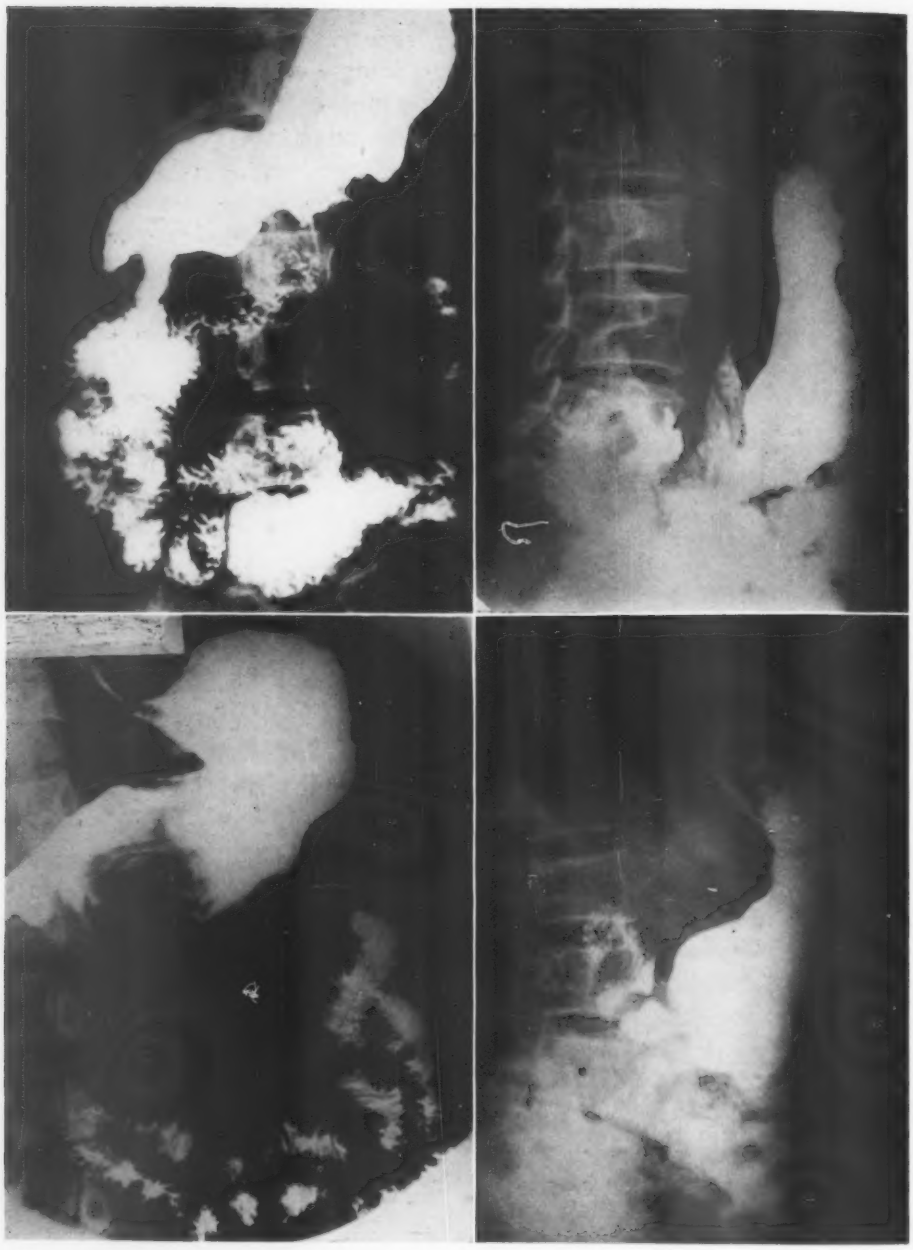

FIG. 7

FIG. 8

FIG. 5.—Hodgkin's disease of the stomach; ulcerating lesion of the greater curvature. Partial gastrectomy was performed. The patient is alive six and a half years after operation.

FIG. 6.—Malignant lymphocytoma. Infiltrating tumor involving cardia and

(*Legend continued on opposite page*)

were Hodgkin's disease or lymphosarcoma (Table I) but there was no apparent variation in gross appearance according to the subgroup of lymphoid tumor. These neoplasms, however, showed gross characteristics differing from leiomyosarcomas in that they were usually larger (smallest 2.0 cm.; largest 18 cm. in greatest diameter) and more likely to grow in the same plane as the gastric wall rather than into the lumen. As a result, the typical appearance was a large, flat mass, usually with extensive, shallow ulceration centrally. In some cases the tumor infiltrated under intact mucosa and smoothed it out or pushed it up into giant rugal folds. While the tumors were often partly necrotic, the non-necrotic portions typically were firm, fleshy and yellow-white. None was encapsulated; most were poorly or irregularly circumscribed. Thirteen of the 32 cases showed metastatic involvement of regional lymph nodes.

Microscopically, invasion of adjacent normal tissue and the usual criteria of malignant lymphoid tumor[17] were present in each case (Figs. 9 and 10). The tumors looked similar to their prototypes arising in other parts of the body. There were no pathologic criteria by which one could specifically tell whether the tumor arose primarily in the stomach or whether the gastric involvement was part of a generalized process.

Distinguishing some undifferentiated carcinomas from lymphoid tumors may give considerable difficulty and it is quite likely that some tumors classified as carcinoma are actually of lymphoid origin. As a rule, examination of multiple sections will reveal areas in which the carcinoma shows some differentiation to allow it to be recognized as of epithelial origin. Another difficulty is in the recognition of malignant lymphoid tumor cells in regional lymph nodes. If the tumor cells are not invading and destroying the lymph node architecture, but are confined to the sinusoids, their recognition may be difficult or impossible.

PATHOGENESIS

The etiology of the sarcomas is unknown. Although the leiomyosarcomas of this series grossly resemble the 21 benign gastric leiomyomas removed during the same period, there is no proof that any of the malignant neoplasms arose from a pre-existing benign tumor. Small, symptomless leiomyomas of the stomach are extremely common findings at autopsy,[13] and it must be conceded that malignant change in such a tumor is a possibility, but difficult to prove. Other than this single factor, there is nothing that can be pointed to as a possible precursor to sarcoma. The gastric mucosa in the uninvolved por-

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media on greater curvature. Total gastrectomy was performed. Patient is alive ten years after operation.

FIG. 7.—Lymphosarcoma. Large infiltrating tumor involving greater curvature of proximal antrum. Partial gastrectomy was performed and roentgen therapy given. Patient is living and well one year and nine months after operation.

FIG. 8.—Lymphosarcoma. Infiltrating tumor involving greater curvature of media and antrum. Partial gastric resection was performed. Patient is alive and well seven years after operation.

FIG. 9

FIG. 10

FIG. 9.—Lymphosarcoma undermining and destroying mucosa. Invasion of underlying muscle (eosin-methylene blue stain, x 12).

FIG. 10.—Lymphosarcoma replacing normal mucosal glands and extensively invading submucosa (eosin-methylene blue stain, x 50).

tion showed no unusual gastritis or other remarkable changes. There was nothing to suggest a relationship between peptic ulcer and sarcoma.

SEX AND AGE DISTRIBUTION

In this series of cases of sarcoma there were 14 females and 27 males, a ratio of 1 to 1.9, which closely parallels the sex incidence in our group of carcinomas of the stomach—females to males 1 to 1.70. In the report of Balfour and McCann there were 13 females and 31 males. On the other hand, some reports indicate an even distribution between the sexes as reported by Cheever and by Douglas. D'Aunoy and Zoeller, in their review of 135 cases, found 43 females and 73 males, a ratio of 1 to 1.7.

It is stated in the literature that sarcoma of the stomach occurs earlier in life than does carcinoma. Balfour and McCann gave the average age for the whole group as 43 years. The average age in this group of 41 patients was 53 years, which is somewhat higher than that in most reports in the literature and very nearly approaches the average age of patients with cancer of the stomach,

TABLE II.—*Age Distribution in 41 Cases of Sarcoma.*

Years	Cases
0–20	0
21–30	3
31–40	5
41–50	8
51–60	13
61–70	7
71–80	5
	41

which we found to be 61 years. The age groups in the series of 41 cases is given in Table II.

In our studies of patients with cancer of the stomach seen in this clinic, 73 per cent were 50 years or older. In this series of patients with sarcomas, 60 per cent were 50 years or older. We believe this tumor is so distributed in all age groups that it cannot be considered a disease of any especial age, although it may be true that large bulky tumors occurring in young people may often prove to be sarcomas. Certainly there are very few distinguishing features so far as sex and age are concerned. The youngest patient in this group was 28 and the oldest 78 years of age.

CLINICAL CHARACTERISTICS

Sarcoma of the stomach presented no characteristic clinical features, and in general the symptoms were similar to those accompanying gastric carcinoma. One feature may be significant, however, in that in general these patients were in good physical condition and did not show the cachexia or anemia so common in many cases of carcinoma of the stomach. In our histories, 33 out of the group of 41 were considered in good or fair condition and only 4 were in poor physical condition.

The average duration of symptoms in this group of cases was ten months,

although symptoms had been present for a period ranging from three weeks to four years. The presenting symptoms were chiefly epigastric pain or simple indigestion and anorexia. In many instances these patients had been treated for varying periods without roentgenologic examination and the presence of a neoplasm in the stomach was not recognized. Twenty-two of the 41 patients had had symptoms for periods of from three months to four years without a definite diagnosis being made. The most common symptoms encountered were vague abdominal distress or pain and anorexia. Pain was epigastric in character, was seldom associated with nausea or vomiting, and was noted in 31 patients. In many cases, food or alkali did give some relief and symptoms were often ulcerlike in character, and this was noted in 19 patients, but this distress was not entirely characteristic of ulcer in the majority either in its periodicity or relief obtained with alkali. Anorexia was also quite common and this was the initial complaint in 27 patients. It was associated with considerable weight loss, which varied from 8 pounds to as much as 60 pounds, and some weight loss was almost a constant finding in every case. Gross bleeding in the form of hematemesis or melena was not a common finding except in the leiomyosarcomas, and in the majority of these tumors it was the most prominent symptom. A history of vomiting blood was noted in 5 out of 9 cases of leiomyosarcoma, and 2 patients had gross blood in stools. Gross blood was noticed once in the stools of patients who had lymphosarcoma, but was usually present in all patients upon laboratory examinations of stools. Anemia was a fairly common finding in the leiomyosarcomas, and hemoglobin concentration varied from a low figure of 30 per cent to as high as 96 per cent. There were 5 cases with a hemoglobin of 75 per cent or less, and 3 with less than 50 per cent. On the other hand, only 5 patients out of 32 with tumors of lymphoid origin had hemoglobin concentrations less than 75 per cent. Determination of the gastric acids was made in 22 of the 41 cases and in 12 patients there was complete absence of free hydrochloric acid. Five patients had free hydrochloric acid of less than 20, and 4 had acid values ranging between 20 and 50.

The presence of high gastric acids does not rule out the presence of malignant tumor of the stomach, but the absence of free acid is particularly significant. Gastric neoplasm should be ruled out by every diagnostic means at our disposal when no free acid is demonstrated, and this appears to apply also to sarcoma.

DIAGNOSIS

Roentgenographic studies were made in all 41 cases of the group. In the group of 9 cases of leiomyosarcoma the diagnosis was leiomyoma in 4 cases; 2 cases were described as benign tumors and in 3 cases the diagnosis was carcinoma. The preoperative diagnosis of sarcoma of the stomach was made in only one case in which the tumor arose from lymphoid tissue, but was suspected in 2 cases. The roentgen diagnosis in 28 was carcinoma, and in one it was benign gastric ulcer.

The roentgenologic examination of tumors arising from lymphoid tissue presents no characteristic picture from which a diagnosis can be made with any degree of certainty. However, as Feldman points out, roentgenologic

examination may furnish valuable information which, together with the clinical data, may strongly suggest the possibility of a sarcoma in the stomach. Feldman emphasized that certain signs are observed in cases of lymphosarcoma which might be helpful, notably (1) a filling defect with smooth margins; (2) a localized type of tumor which is round and smooth; (3) in the diffuse type, involvement of large portions of stomach simulating linitis plastica; (4) relief pictures of the stomach often show mucosal rugae in thick folds; (5) the presence of palpable tumors in young individuals strongly suggests the possibility of sarcoma and (6) the presence of multiple ulcers suggests it. On the other hand, it is well to point out that the roentgen examination may not reveal evidence of any definite abnormality in the stomach, and this was true in two cases.

Holmes, Dresser and Camp, in a discussion of the roentgenologic characteristics, also stated that there is nothing diagnostic in the roentgen appearance of the stomach, although peristalsis is not interfered with as much as in most cases of carcinoma. Leiomyomas, however, may have a more characteristic roentgenologic appearance; they very often are diagnosed as benign polypoid tumors of the stomach, and present a circumscribed globular filling defect in the stomach which is fairly characteristic.

Gastroscopic examination may confirm the presence of tumor. Again there are no definite gross features upon visualization of the tumor which will establish the diagnosis of sarcoma, and in the majority of cases a definite diagnosis can be established only by laparotomy.

Gastroscopic examination, however, should be done in all cases in which gastric sarcoma is suspected and Schindler *et al.* stated that more experience in gastroscopy and better diagnostic methods might well reveal a higher incidence and a higher preoperative diagnosis. Taylor in a review of 152 cases in the literature stated that in those cases in which gastroscopy was done there was no characteristic diagnostic feature that could be recognized.

Palpable abdominal tumors were demonstrated in 11 patients, and again it is important to point out that this bore no direct relationship to resectability or to prognosis and unless there was evidence of widespread tumor, exploratory laparotomy should be carried out in all these cases. In this series all but 4 proved to be resectable either by partial or total gastrectomy.

TREATMENT

The only treatment of value in these cases of gastric sarcoma is radical surgery which, in the case of tumors arising on lymphoid tissue, should be followed by deep irradiation treatment. Inasmuch as the majority of these cases are not usually recognized until the time of surgical intervention it would be useless to employ deep irradiation preoperatively, since irradiation treatment would have no effect whatever on gastric carcinoma, and valuable time might be lost before surgical extirpation of a sarcoma could be carried out. Holmes *et al.* suggested resection whenever possible before irradiation. Irradiation of a hollow viscus containing large ulcerating tumor masses always entails danger of gross hemorrhage and perforation. It would also seem wise

in the surgical exploration of extensive tumors arising in the stomach, and which are considered nonresectable, that a biopsy specimen be obtained in every instance so that these infrequent cases of sarcoma could be recognized and irradiation treatment employed if radical surgery does not appear possible.

Only eight patients had postoperative treatment and in seven this was done because of recurrence of tumor; the eighth patient upon surgical exploration and biopsy proved to have Hodgkin's disease of the stomach which was too extensive to be resected. Following operation he was given roentgen therapy over the abdomen and is alive five and one half years after exploration but now has Hodgkin's disease involving the stomach, abdomen, mediastinum and cervical lymph nodes. After roentgen therapy he was able to eat well and remained in reasonably good health until recently.

TABLE III.—*Operations for Sarcoma of the Stomach (41 Cases).*

Operation	Leiomyosarcoma	Lymphoid Tumors
Exploration and biopsy	0	4
Excision	2	0
Partial gastrectomy	5	19
Total gastrectomy	2	9
Total cases	9	32

It is our impression that postoperative irradiation should be employed routinely in all lymphoid tumors and this might well increase the five-year survival rate and serve to prolong life. Whether irradiation prolonged life in the seven patients who had operation and postoperative irradiation is difficult to state with any degree of certainty; two lived six years, one lived two years and four lived one year, but roentgen treatment was used, as already stated, only in seven patients with recurrent tumor appearing after operation, except in the one case in which resection was not thought feasible.

No irradiation treatment was used in those patients operated on for leiomyosarcoma. Surgical treatment in the entire group of gastric sarcoma consisted of removal of the tumor by partial gastric resection, which was done in 24 cases, and total gastrectomy, which was done in 11 cases. In 4 patients the tumors were not resectable and exploration and biopsy was done. Two of the leiomyosarcomas were removed by excision only of the tumor mass (Table III). There were two postoperative deaths in 41 cases, an operative mortality of 4.9 per cent.

RESULTS OF TREATMENT

The follow-up results of the patients operated on for leiomyosarcoma are surprisingly good (Table IV). Eight of the nine patients are alive and well from one year to 20 years; six patients lived five years or longer, in other words, a 67 per cent five-year or more survival after operation. One patient is dead six years after partial resection, the cause of death unknown (Table IV).

Two patients with leiomyosarcoma had total gastrectomy and are alive and well 12 years and two years respectively. In two patients local excision of the tumor only was done because resection would have involved total gastrectomy.

Both patients are alive and without recurrence, one ten years (Fig. 1) and one two years (Fig. 2) after operation. It may be true that when wide local excision is possible this may be sufficient in view of the low grade malignancy of this tumor.

TABLE IV.—*Leiomyosarcoma—Nine Cases.*

No. of Years Living and Well	Operation	Cases
20	Partial gastrectomy	1
12	Total gastrectomy	1
10	Transgastric excision	1
10	Partial gastrectomy	1
6	Partial gastrectomy	1
5	Partial gastrectomy	1
2	Partial gastrectomy	1
2	Total gastrectomy	1

One patient is dead (cause unknown) more than six years after partial gastrectomy. No operative mortality.

Of those patients who had tumors developing from lymphoid tissue, two died immediately after operation and 12 lived an average period of 13 months (Table V), varying from six months to two and one half years. Of the 32 patients with tumors of lymphoid origin, 18 (56 per cent) are alive and well (Table VI). Twelve patients have lived five years or longer, five of whom had Hodgkin's disease; six patients are alive and well six months to two

TABLE V.—*Malignant Tumors of Lymphoid Origin (14 Dead).*

Length of Life After Operation	Hodgkin's Disease	Other Lymphoid Tumors
Operative death	1	1
6 months	1	3
1 year	2	2
1½ years	1	0
2 years	0	1
2½ years	0	2
Total	5	9

years. The prognosis on the whole is very much better than it is with carcinoma. It is surprising to note that five patients with localized Hodgkin's disease are alive five years or longer, whereas four patients with Hodgkin's disease died within the first year. Four patients with Hodgkin's disease lived five years or more and one patient is living and well ten years following resection. One patient with Hodgkin's disease has survived five years with roentgen therapy. Excluding the group of patients with Hodgkin's disease, seven of the 22 are living and well over five years, one each five, six, seven, eight years and three for nine years. Eighteen patients or 44 per cent of the group of 41 patients with sarcoma have survived five years or more, which represents a much higher five-year survival rate than in carcinoma of the stomach.

It is of interest to note that 11 patients had total gastrectomy for removal of their sarcoma; of this group, five (45 per cent) have survived five years or longer. Of a group of 26 patients whose tumors (except in 2 cases) were

removed by partial gastric resection, 12 lived five years or longer, a five-year survival of 46 per cent. If one excludes the leiomyosarcomas and considers only the more malignant lymphoid tumors the five-year survival following total gastrectomy was 33 per cent, whereas the five-year survival rate follow-

TABLE VI.—*Malignant Tumors of Lymphoid Origin; 17 Patients Alive and With No Recurrence**

Length of Life After Operation	Hodgkin's Disease	Other Lymphoid Tumors
6 months	0	2
1 year	0	2
1½ years	0	1
2 years	0	1
3 years	0	0
5 years	1	1
6 years	2	1
7 years	0	1
8 years	0	1
9 years	0	3
10 years	1	0
Total	4	13

* One patient with Hodgkin's disease was not operated on but was given irradiation treatment and is alive five and a half years later but has generalized Hodgkin's disease, making a total of 18 patients still alive.

ing partial resection was 42 per cent. It must be remembered, however, that total gastrectomy was done only for the more extensively involved cases.

It is not surprising that the survival rate following resection of leiomyosarcoma is high. The tumors appeared to be adequately excised locally and there were no demonstrable metastases at the time of surgery. Only one patient died and the cause of death in this case is not known.

The survival rate following resection of malignant lymphoid tumors of the stomach is also surprisingly high. This is in marked contrast to the low survival rate following resection for gastric cancer. The patients still alive had tumors which showed no apparent difference in regard to size, location, ulceration or metastases from the tumors in the patients now dead. Seven of the 17 patients still alive showed tumor in the regional lymph nodes in the resected specimen. One, for example, with Hodgkin's disease had a 10 cm. gastric tumor and metastases to each of 15 curvature lymph nodes, but the patient is alive and without evidence of recurrent disease six years following resection (Fig. 5). The remarkable survival rate in the lymphoma series does not, of course, necessarily mean a cure, for lymphoid tumors in other portions of the body may be present for a number of years before causing death. However, this is unusual and it is significant to note that Nathanson and Welch found that the median life expectancy for patients with generalized lymphoid tumors was two years.

SUMMARY

A clinical analysis is given of 41 patients with sarcoma of the stomach treated in the Lahey Clinic over a period of 20 years, an incidence of sarcoma

of the stomach of 3.7 per cent. The diagnosis of sarcoma is rarely made before surgical intervention and the clinical picture of this disease is very little different from that of carcinoma. The outstanding symptoms are anorexia, indigestion, epigastric pain and weight loss; cachexia is uncommon.

There were two types of gastric sarcoma in the series, those of smooth muscle origin, the leiomyosarcomas, and the malignant lymphoid tumors.

Roentgenologic examination presents no characteristic picture which permits recognition preoperatively except in the cases of leiomyosarcoma. Certain roentgenologic findings suggest its possibility, and sarcoma should always be considered in the diagnosis.

The prognosis is surprisingly good after surgical treatment; 18 patients or 44 per cent survived five years or longer.

Treatment of gastric sarcoma is radical surgery. Irradiation treatment should be used postoperatively for patients whose tumors arise from lymphoid tissue. There were two deaths following operation in 41 patients.

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THE LONGMIRE OPERATION FOR COMMON DUCT OBSTRUCTION*

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FROM THE LEXINGTON CLINIC, LEXINGTON

IN 1948 LONGMIRE AND SANFORD reported a procedure for "treating extensive obstruction of the common duct or common hepatic duct by anastomosing one of the intrahepatic biliary ducts to the jejunum following partial resection of the left lobe of the liver." They pointed out that this method might be of great value where the extra-hepatic ducts had been largely destroyed by benign disease or "used up" by previous ineffective operations. Because of the dense adhesions usually present in the right upper abdomen the authors mobilized the left lobe of the liver, exactly as most surgeons do in abdominal vagotomy.

The procedure then is outlined. A broad gauze band or tape is placed around this left lobe close to the round ligament and this is pulled forward until the entire lobe is accessible to surgery. Incision is then made into the left lobe close to the edge of the tape in a sagittal plane. Bleeding from the liver is controlled by manual compression of the medial portion of the left lobe and by carefully placed mattress sutures in the lateral portion. Later, mattress sutures in the medial portion replace the hand compression. These sutures may or may not be tied around blocks of Gelfoam. The liver is penetrated in this way until a bile duct of sufficient size is encountered so that anastomosis will be easy and a good flow of bile will be ensured. The authors[2] state that such a major duct may "lie in the inferior or caudal half of the substance of the left hepatic lobe, at times quite near the inferior surface."

A loop of jejunum is then drawn up and sutured to the posterior inferior edge of the liver, an opening is made into the jejunum at the proper point. A careful mucosa-to-mucosa suture is made between the end of the duct which has been freed by cutting away or curetting the liver tissue immediately surrounding it and the opening in the jejunum. The jejunum is then sutured firmly to the anterior superior edge capsule, thus covering all the raw liver area. The anastomosis is made around a catheter which is held in place by catgut suture for days or weeks. A proximal entero-enterostomy is then done or the afferent loop is interrupted and a Roux type of anastomosis is made.

Longmire and Sanford[2] have now reported four successful instances in which they have used this method for adults with benign obstruction of the extra-hepatic biliary system. They call attention[1] to their own work and that of others which shows that the operation will be most effective in those whose biliary system block is below the junction of the left and right hepatic ducts.

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 7, 1949.

Fortunately this is true in most instances so that bile from the larger right lobe flows down the right hepatic duct until it meets the obstruction and then backs up into the left hepatic duct, reversing the flow in this duct until it flows out into the intestine through the left lobe duct which has been joined to the jejunum. In the limited work which has been done in this field no biliary connection has been found between the right and left lobes of the liver except through the main ducts in their junction to form the common duct. The exception seems to be that both right and left duct systems drain the caudate lobe,[1] so that even if there is no direct connection between right and left hepatic ducts this common drainage of the caudate lobe would probably be sufficient to maintain adequate liver function. Even if there were no connection between right and left lobes the authors[2] state that their experience would suggest that partial hepatectomy and cholangio-jejunostomy be considered because of probable compensatory hypertrophy of the remaining part of the left lobe with atrophy of the right lobe. In support of their contention they cite the experience of Cattell quoted by Lahey.[3]

Since the first report of this ingenious method others[4, 5] have reported its successful use.

The patient whose case history follows seemed to us suitable by all criteria for the procedure which has now become known as the "Longmire operation."

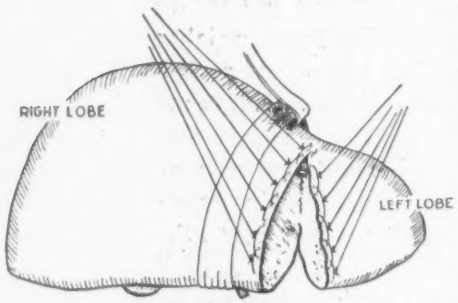
CASE HISTORY

Mrs. E. V., No. 107–268, age 46, was admitted to St. Joseph's Hospital, Lexington, Kentucky, July 27, 1946. She had been operated on five weeks previously elsewhere and the gallbladder, containing one stone, was removed. Prior to this cholecystectomy she had not been jaundiced. Three days after the operation she became jaundiced and had daily fever to 101 degrees and was dismissed from the hospital on the eighteenth postoperative day, still jaundiced and still quite ill. When she first was committed to our care two and a half weeks later she was an acutely and gravely ill woman, dehydrated, jaundiced, vomiting, and complaining of pain in lower right chest. On admission her temperature was 102, leukocytes 18,000 and icterus index 60. After necessary preparation the abdomen was explored on the fourth day after admission, going through the recently healed wound in the right upper area of the abdomen. A large amount of pus and bile was encountered in this area and no structures were identified. The abscess was drained and from this she made a slow recovery and left the hospital 4 weeks after the operation with the wound healed, icterus index of 12 and feeling well.

She returned several times for observation and was improving until September 9, 1946, when she again became jaundiced and again had low grade fever (no chills) and nausea with upper abdominal pain. This condition alternated with periods characterized by relief of pain and jaundice and return of color to the stools. She was admitted again to hospital November 4, 1946, with jaundice, acholic stools and pain and was operated on again November 9, 1946, when a large (500 cc.) collection of bile-stained thin fluid was evacuated from a pocket encapsulated between the lesser curvature of the stomach and the liver. There was still no opportunity to explore the gallbladder area or common duct. Again she made a slow recovery and left the hospital on November 18, 1946, with jaundice clearing (icterus index had fallen from 115 to 60) and the stools brown. Roentgenograms made by injecting an opaque medium through a catheter left in the pocket at the time of operation showed no biliary connection. During the next 12 months she was never free from spells of jaundice, chills and malaise for more than 3 months at a time, though her general condition was slowly improving.

She was again admitted to St. Joseph's Hospital February 20, 1948. Though once more jaundiced, with index of 61, her liver function was good. Cephalin flocculation was 2 plus, serum protein 6.5 with albumin 4.3 and globulin 2.2. The prothrombin time showed a satisfactory response to vitamin K. The blood count showed red blood cells 4.5 million, hemoglobin 13.5 Gm. per 100 cc. and for the first time we thought she was in condition for a careful exploration. This was carried out February 24. The common duct was found after difficult dissection of very dense and vascular adhesions between the liver and duodenum. It was greatly thickened, white and only moderately dilated. It was opened and dark, not "gold," bile flowed freely. A small sound was easily passed into right and left hepatic ducts but half an inch below the opening there seemed to be complete fusion of the duct walls. The duct was followed down by gauze dissection for two and a half inches and it felt hard, thickened and not enlarged. Hope of finding a relatively normal lower end of the duct was abandoned and an anastomosis was made between the opening already made and the adjacent duodenum. She improved rapidly and left the hospital on the twenty-second postoperative day.

FIG. 1



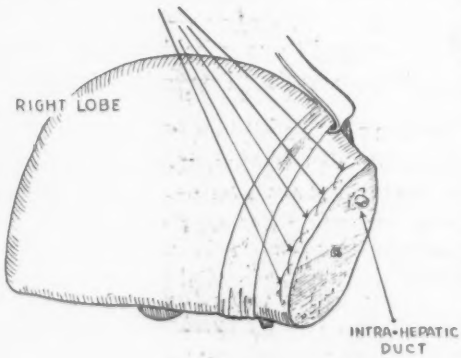


FIG. 2

FIG. 1.—Diagram shows an incision into the left lobe. Mattress sutures placed to control bleeding have been left long for traction. A bile duct has been encountered and cut. It was too small for satisfactory anastomosis.

FIG. 2.—The lateral two-thirds of the left lobe completely removed. A large bile duct has been found near the posterior edge of lobe. Anastomosis between a loop of jejunum and this duct has been made.

She then gained weight and felt well "for the first time in two years." Three months later, however, she had a mild spell of malaise, pale stools and faintly yellow skin, which cleared up in two or three weeks.

In October, 1948, she was again a victim of chills, fever, jaundice and malaise, and these episodes continued until they again became severe in May, 1949, and she was re-admitted to hospital June 8, 1949. Because of the dense adhesions and free bleeding at the time when the choledocho-duodenostomy was made, the benign nature of the obstruction, and the pitiable condition of the patient, an intrahepatic duct jejunal anastomosis was done on June 18, 1949, after 10 days preparation. The operation was carried out as nearly as possible according to the description of the various authors.[1, 2, 4, 5] One mistake in technic we will not make again, should opportunity ever present, is using the mattress suture ends to retract the two portions of the left lobe of the liver (Fig. 1). After 20 or 30 minutes of this they cut through in part, bleeding was profuse, and the sutures had to be replaced frequently. Also we did not tie them around Gelfoam which would have been wiser to do. We omitted this because Gelfoam is soft and "messy." She received 2500 cc. of blood during the operation which lasted two hours and a half.

A careful mucosa-to-mucosa suture of hepatic duct to jejunal loop was done with 000 catgut. A duct sufficiently large to use was not found until the posterior-inferior

border of the left lobe was almost reached (Fig. 2). The loop was fixed to the liver capsule with interrupted sutures of fine silk and an entero-enterostomy was done well away from the duct anastomosis. The duct jejunal anastomosis was made around a rubber catheter (size 16) and the catheter was brought out of the distal loop of the jejunum just distal to the entero-enterostomy. The Witzel method was used in bringing the catheter out of the bowel. In this way bile output and cholangiograms could be

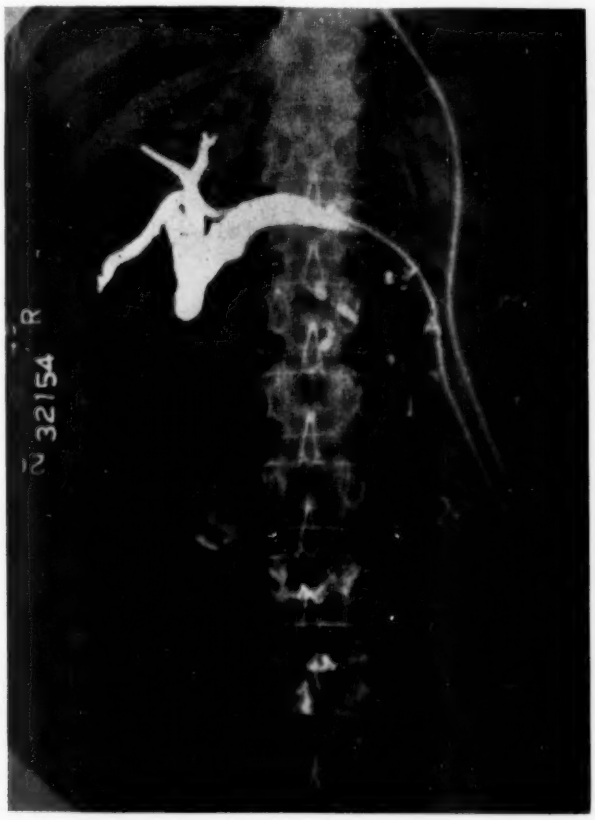


FIG. 3.—This shows cholangiogram 18 days after operation. A catheter introduced into the hepatic duct of the stump of the left lobe passing through the anastomosis of his duct to the jejunum was brought out through the jejunum and abdominal wall. It shows a dilated left hepatic duct, its junction with the right hepatic duct, the obstruction just below this junction in the common duct and the dilated right hepatic duct and main bile radicals of the right lobe.

No connection between biliary systems of the right and left liver lobes could be found.

studied at leisure. During the first 24 hours 500 cc. of bile, first black then gold, drained into the bottle to which the catheter was attached. Her daily output of bile was good, she improved steadily, without fever after the third day, and on the fifth day after operation the stool by enema showed some acholic feces (old) and some brown feces. There was no drainage from the wound. On July 6, 1949, 18 days after operation the patient was in good condition, bowels moving well and the feces were normal in

color. On this day cholangiograms were made using diodrast as the opaque medium (Fig. 3). This shows the catheter in the left hepatic duct, a complete obstruction just below the junction of the left and right hepatic ducts, probably at the site of the former choledochoduodenostomy done 16 months previously. The roentgen ray study shows that the right intrahepatic ducts are dilated. No connection could be seen between the biliary systems of the right and left lobes of the liver except through the main hepatic ducts. This supports the experimental and postmortem findings of other observers. These cholangiograms are very similar to those of Walters[6] shown in his discussion of Longmire and Sanford's[2] most recent article.

She continued to do well. The catheter was removed after roentgen ray study was made on July 9, 1949, which showed only a little of the opaque medium left in the bile canals of the right lobe. The abdominal wound did not drain, and she was dismissed from hospital July 11, 1949, with an icterus index of 50.

On September 15, 1949, she was examined in the office, her weight was 97 pounds (a gain of 10 pounds), her stools were brown, and she felt quite well. The icterus index was 12, the hemoglobin was 13 Gm. per 100, and she had no pain or symptoms of dyspepsia.

Once more, in November, 1949, she had an episode of chill, fever 102, jaundice, icterus index 53, and was kept in hospital under observation 8 days. The stools were acholic streaked with brown. After she returned home she had copious brown stools, diarrhea, fever 101, and felt better than at the time of her last dismissal.

DISCUSSION

Longmire and Sanford have devised an ingenious operative procedure for draining the bile into the intestinal tract when the common bile duct has suffered a benign obstruction and where repeated ineffectual attempts have been made to re-establish the drainage. It is well to point out here the destructive action which pockets of extravasated bile have upon the common bile duct. While our series of complete constrictions of the common duct following biliary tract surgery is small, of the six seen and operated on by us in the last two years, four have given a history of extravasated bile. We believe this was a major factor in producing the thickened fibrosed duct in the patient described in this report. We think it possible that accumulated bile may be a factor in causing all common duct strictures where the obstruction extends for more than one cm. along the course of the duct. If this be true, there is ample reason for placing a drain in Morrison's pouch in every patient operated on for biliary tract disease. It makes little difference where the drain is brought out, through the incision or, in case of longitudinal incision, through a lateral stab wound. The prognosis of all the patients so far reported who have suffered this operation has been satisfactory, though our patient has been operated on too recently to pass judgment.

Our patient returned with jaundice, chills and fever November 15, 1949. She recovered, but this bodes ill for her future. Should we be forced to again subject her to surgery we shall probably attempt to re-open her intrahepatic duct stoma rather than attack the dense adhesions in the right upper quarter of the abdomen. We think our incomplete success and possible failure should not be charged to the operation. It may well be due to failure of the operator, or individual reaction (fibrosis formation) on the part of the patient.

The operation technically is not easy but if the patient is properly pre-

pared and the procedure carried out slowly and painstakingly it is easier for us than mobilization of the duodenum and dissection of the lower end of the common duct, perhaps in the pancreas, as advocated by Lahey and Cattell. We think the catheter through the anastomosis may be brought out of the jejunum and attached to a bottle without risk or injury to the patient. It furnishes a convenient way to study the intrahepatic ducts, the nature of their connections, and easily establishes in any given case whether or not there is any connection between the biliary systems of the right and left lobes of the liver other than through the main hepatic ducts. It determines where the obstruction is in relation to the junction of these main ducts and if this obstruction is distal to their junction, as it seems to be in most cases, the outlook should be favorable.

It would perhaps be wiser to make cholangiograms before the operator has removed the left lobe. In our case and in the case reported by Sanders[4] the left lobe was removed before cholangiograms were made. If the obstruction is found by roentgenogram to extend upward and include the right and left main ducts so that there is no biliary connection between the right and left lobes, then certainly it would be safer to preserve as much of the left lobe as possible.

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DISCUSSION.—DR. FRANK H. LAHEY, Boston: I never mean to discuss this operation in terms of discouragement because it is another procedure which we can have available in these cases, and they are complicated enough so that we need as many methods of dealing with them as possible. In addition, we must always give Doctor Longmire every credit for his ingenuity in working this operation out.

I always speak of numbers with hesitation, but bile duct injuries are something with which, up to recently, no one has had a large experience; therefore from our good sized experience I would like to present some conclusions in an attempt to help others.

We have now operated on 280 patients for bile duct strictures. This is a serious criticism of surgery. If we have operated on that number of patients with duct injuries, how many others have died? How many others have been operated on by other surgeons? Think of how many gallbladder operations are performed daily, and that every cholecystectomy exposes the patient to this hazard. We have to realize that every one of these patients who has had a safe removal of the gallbladder is well, that every one with an injury to the bile ducts is an invalid, and that every procedure you employ after that is a makeshift one.

I do not report 280 cases of bile duct injuries out of any pride. They are a headache for everybody. The patient is exhausted from every point of view. His family is exhausted, and before he gets through the surgeon has exhausted nearly every resource he possesses.

What we try to do, of course, is to attempt to establish a makeshift procedure which will enable some of them to live and will prolong the lives of others.

There are some basic things about which we must be careful if we make use of the Longmire procedure. The same principles must be employed as with anastomosis of the cut end of the hepatic duct to the duodenum or the jejunum. You cannot implant a tube tied into the end of this duct into the jejunum or into the duodenum. Doctor W. J. Mayo, a long time ago, demonstrated something which still is very sound; that is, you must do mucosa-to-mucosa anastomosis in bile duct anastomoses with the intestinal canal, and the aperture must be a large one if patency is to be maintained. In addition, we must not interpret any operation as successful short of two years, because no matter what is done in the way of anastomosis, even mucosa-to-mucosa anastomosis, there is a high incidence of closure, and any operation in which a wide mucosal anastomosis has not been done is almost sure to close. When hepatico-intestinal anastomosis is undertaken, the ones which will do well will be those with drumstick ends of the hepatic duct. Those are the patients who have had an obstruction for some time and have well dilated ends of the hepatic duct, so that a really accurate type of anastomosis can be done.

In discussing the Longmire precedure previously, I have called attention to the fact that, for this operation to function and really drain the entire liver, there must exist an uninjured common hepatic duct, and have repeatedly stated that when such a common duct does exist, it is in most cases possible to do a quite accurate mucosa-to-mucosa hepaticojejunostomy without sacrificing any liver tissue. One must remember that when only the left hepatic duct exists, a considerable portion of the only functioning portion of the liver, that is the left lobe, must, in the Longmire operation, be sacrificed in an attempt to find a left hepatic duct of such caliber as to be useful.

Doctor Cattell will soon publish his experiences with ten patients in whom anastomoses were made solely between the common duct and the left hepatic duct, the right having been destroyed. I have just operated on one of these patients in whom, following previous surgery elsewhere, there was complete atrophy of the right lobe of the liver, the left lobe becoming as large as a normal right lobe. It is for this reason that Doctor Massie's warning that one must be careful not to destroy the left lobe of the liver when this operation is done is extremely important.

What can we offer in the way of end results in any of these 280 patients upon whom we performed anastomosis? We have just completed a follow-up study on 229 of these patients. We have discarded all cases that have not been followed for two years and ten that were lost to the follow-up, and that is the difference in the figures 229 and 280—the total number of patients. I have just reported these figures at the International Surgical Society. One important thing that must be considered in these operations is the economic loss, time loss and suffering. In 239 patients there were 761 operations, either before coming to us or after coming to us. The mortality in this group has impressed us; it was 22 per cent prior to 1939; it is now, from 1940 to 1948, down to reasonable figures —patient mortality 6.5 per cent and operative mortality 4.4 per cent. We have now done this operation in 71 cases with one death in the last three years. One thing this experience has taught us is something that we already knew, but these trying cases have impressed it upon us even more forcibly, that is, that there is no substitute for experience. You have to suffer the headaches with these operations to learn what can be done and what cannot be done.

Let us take the end-to-end anastomoses in which only a small section of duct was cut out; 78 per cent of those patients have good or fair results; all of them were operated on more than two years ago. It is interesting that if one interprets these when only one operation has been done, only 60 per cent obtained good results, but if you re-operate upon some of the failures you can add another 20 per cent good results. One must persist and not be discouraged with these cases if the first attempt is not successful.

In patients who have had the duct dissected out of the pancreas and direct end-to-end anastomosis, as Doctor Cattell and I have reported, 73 per cent have had good results.

In patients who have had the short crush type of strictures, in whom plastic operations of the Heineke-Mikulicz type could be done, 100 per cent have had good results; those are the easy cases to do.

Of those who have had implantations of the tube to bridge defects, only 20 per cent have had good results.

It is interesting and encouraging that we used to be dubious about the results of anastomosing the intestine to the hepatic duct. It is proving, however, quite a good operation; 73 per cent of these cases have had good results.

One final word about these cases: The best time to repair them is fairly close to the time the ducts are injured before scarring and fibrosis have taken place. Whenever an unsuccessful attempt is made at repair, the next operation will be made more difficult.

DR. HARWELL WILSON, Memphis: I think, as both Doctor Massie and Doctor Lahey have emphasized, that the follow-up reports of all these cases, regardless of the method by which they are treated, are exceedingly important. Since at the meeting of this Association one year ago we reported upon our experience with the Longmire operation, I think it opportune to give a brief follow-up report at this time. We have recently examined the patient upon whom we performed cholangiojejunostomy with partial hepatectomy in March, 1948. This individual who, at the time of operation, we all felt would certainly be dead within a short time unless continuity of the biliary tract could be re-established, has, since the operation, worked most of the time at his occupation as a stationary engineer, except for some time spent on a pleasure trip to California. Bile has been present in the stools continuously since operation.

During this time the patient has not been entirely well. He has shown evidence at times of mild hepatitis. At the time of our original report we pointed out that microscopic examination of the liver tissue removed at operation and also liver function tests performed showed evidence of hepatitis. He has nevertheless been able to work almost continuously. I would like to show one slide to point out briefly two technical considerations which I feel may be helpful. We employed a modification of the originally described operation, utilizing a Roux-Y loop in order to accomplish an end-to-end anastomosis between the left intrahepatic duct and the jejunum. The duct was sutured to the mucosa of the end of the jejunum with fine interrupted sutures. A small catheter had been previously placed in the duct and the catheter was brought outside the jejunum below the site of anastomosis by utilization of the Witzel method.

The second technical point I wish to emphasize is to call attention to the artery which usually accompanies the duct. This artery must be ligated. It is important that the through-and-through sutures used to control bleeding from the liver edge do not include the duct. I certainly agree that in a case where we can perform an end-to-end anastomosis of the common bile duct this should be done and I think it unlikely that any one individual will see many cases where the Longmire operation is indicated, since usually other methods which are more suitable can be used. Nevertheless, just as it behooves all surgeons who deal with arteriovenous fistulas to know all methods so as to be able to deal with a given case, it is also true that the surgeon dealing with the biliary tract should be familiar with all useful procedures.

DR. FRANCIS M. MASSIE, Lexington, Ky. (closing): I think the presentation of this paper would be justified if only for the fact that it has evoked the discussion of Dr. Lahey and Dr. Wilson.

There is just one other point I would like to bring out and that is that extravasations of bile which are pocketed in the area of the common duct may be the cause of complete strictures of the common duct. Within the past two years I have operated on six patients with stricture of the common duct and four of the six had accumulations of bile, mostly infected of course, for prolonged periods from one week to months, outside the common duct. I believe that must be a factor in this type of occlusion of the duct. It is not, properly speaking, a stricture; it is an occlusion for a distance, in one instance, of four and a half inches.

SURGICAL ASPIRATION OF THE BOWEL IN ADVANCED OBSTRUCTION*

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AMONG THE MANY PROBLEMS to be solved in dealing with patients suffering from acute obstruction of the small bowel none is more important than that of successfully dealing with the great distention present in late cases. In early cases which may be relieved by suction through the stomach or by operative correction of the obstructing lesion great distention does not exist. In the late cases, however, there has been grave damage to the patient by the loss of fluid, electrolytes and protein from the blood stream into the intestine and grave damage to the bowel itself by increase of intraluminal pressure. Uncorrected, the former leads to shock and the latter to ulceration, necrosis and perforation of the bowel wall.

The blood circulation in the wall of the small bowel has been beautifully presented by Noer, Derr and Johnston.[10] They say "The ability to prevent filling (of the small blood vessels) by marked distention was no surprise. The degree of vascular interference caused by moderate degrees of distention, however, was greater than anticipated. This finding emphasizes the need for complete intestinal decompression in all conditions associated with intestinal distention. Further, the results indicate a need for constant decompression wherever the integrity of the intestine is threatened by injury or operative trauma."

Van Zwalenburg[14] in 1932 emphasized the importance of collapse of the veins in the wall of the intestine by pressure from great distention; this not only impairing the circulation but increasing the outpouring of fluid and electrolytes into the intestinal canal.

The importance of great distention when not corrected as a factor leading to death of the patient has been recognized for many years. Nicolas Senn[12] wrote 60 years ago advising that it be corrected at the time of operation and its recurrence prevented. To accomplish this he advised that the bowel be opened and aspirated. In order to empty loops kinked by the weight of the contents, he manually straightened them to encourage the liquid material to flow toward the point of aspiration. He further advised, after the obstructing lesion had been corrected, that pressure dressings and saline cathartics be used to prevent recurrence of the stasis and distention. J. M. T. Finney, Jr.,[4] recognized the same danger to patients having peritonitis from ruptured appendicitis

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 7, 1949.

and advised the use of castor oil after the appendix had been removed. This striking departure from old rules has been used in our hospitals with great satisfaction. This was reported by Lee[7] in 1946.

Moynihan[9] in 1926 advised enterotomy and aspiration. He said that six feet of bowel could be collapsed on a six inch tube. Harvey Stone[13] in 1926, from experimental and clinical evidence, felt that a toxin was developed in the bowel contents which, when absorbed as the circulation improved, might cause a fatal intoxication. He advised that the obstruction be relieved as soon as possible and the contents of the intestine removed at the time of operation.

Holden[6] in 1930 and Cheever[1] in 1932 have reported desperate cases who recovered following operation where the bowel was emptied and the obstruction relieved. Elman[3] in 1934 suggested that by removal of the bowel contents the circulation becomes so improved that the toxic substance is rapidly absorbed. He thought that this might explain certain deaths after operation.

Among others Morton[8] in 1932 and Ochsner and Storck[11] in 1936 condemned handling of the distended bowel and efforts to empty it. Their objections were based largely on experimental work but also on clinical observations and references to the literature.

Wangensteen[15] in 1942, again in 1947[16] and Dennis[2] in 1949 have advocated aseptic suction of the distended bowel in order to expose the obstructing lesion. They do not stress the importance of emptying the bowel to restore its function.

Harper and Lemner[5] in 1946 stress the damage done to the bowel wall by great distention, such as ulceration and later perforation, and suggest that bacterial infection in the damaged portions may lead to fatal intoxication.

We were greatly impressed several years ago by the recovery of a patient after a grave operative accident. This 40-year-old woman entered the hospital with the history of obstruction which had been complete for a week. Her general condition was remarkably good. On exploration, complete obstruction of the transverse colon by an annular carcinoma was found. The ileocecal valve was entirely competent, so that enormous distention of the right colon had resulted; the small bowel was only moderately distended. The distended colon was grayish green in color and the wall was very thin. With the most gentle handling of the bowel, effort was made to isolate this vulnerable bowel by packing. The cecum ruptured and spilled its contents into the peritoneal cavity. Through this opening the bowel was completely emptied by suction and immediately there was marked improvement in the bowel wall. It was then possible to securely suture the bowel around a large tube. The peritoneal cavity was cleansed by copious irrigation and suction. The patient made a remarkably prompt and uncomplicated recovery, and later the transverse colon lesion was successfully resected. This happy outcome resulted, we are sure, from the restoration of the bowel wall by decompression and in spite of the gross soiling of the peritoneal cavity.

We have had a small series of cases of bowel obstruction of long standing resulting in great distention of the small bowel with the usual grave chemical

changes in the blood stream and poor condition of the bowel wall. At operation the small bowel has been emptied as completely as possible by aspiration, an enterostomy established, and the obstructing lesion removed. The recovery of these desperately ill patients has been remarkable.

The method used has been simple. A distended loop of bowel is emptied by gentle manipulation and compressed by fingers; clamps are not used because the bowel is usually in poor condition. A pursestring suture is placed, an opening is made into the lumen through this pursestring, an ordinary suction tip is inserted, and the suture is tied around it. This suction tip is six to eight inches long. Low pressure suction is then started, and as the bowel collapses it is threaded on the suction tip. About six or eight feet of bowel can thus be emptied, and by gentle manipulation other distended coils above can be straightened out and the fluid and gas contents guided toward the suction. We have tried a small rectal tube with large perforations, but find that it draws the mucous membrane into the openings when the bowel collapses. We have had much better results with the conventional suction which is applied inside the perforated sheath. If necessary, the direction of the suction tip can be reversed to empty the bowel in the opposite direction. In some cases two openings were made. We have removed from 2000 to 5000 cc. of fluid from the small bowel in these cases and, of course, a considerable amount of gas. When the aspiration has been completed, an enterostomy is established through the same opening. The bowel wall rapidly improves when the distention is relieved. Appropriate steps are then taken to relieve the obstructing lesion.

CASE REPORTS

Case 1.—*Internal Hernia.* This 17-year-old girl was admitted to the Medical College of Virginia Hospital from another hospital, where her appendix had been removed 10 days before on account of abdominal pain of 2 days duration. She had become progressively more distended and when admitted was in poor condition. The blood N.P.N. was 100 and chlorides 437. She was given large amounts of glucose and saline intravenously and 2 transfusions of blood. At operation a coil of distended terminal ileum was adherent to the old incision. In separating this loop the bowel was ruptured and a considerable amount of contents spilled. The obstructing lesion was a large sac in the upper abdomen, with a small opening having a very thick bandlike edge. The suction tip was placed through the laceration and the bowel emptied and at the same time delivered from the sac. Three thousand cc. of fluid were thus removed. A catheter enterostomy was made through the same opening. The sac was almost completely removed. The patient did not become distended after operation but developed a pelvic abscess which was drained. Her recovery was then uneventful.

Case 2.—*Paralytic Ileus.* Medical College of Virginia Hospital. This 24-year-old woman had become progressively more distended for 4 days following a cesarean section. X-ray examination confirmed the clinical impression of mechanical small bowel obstruction. At operation great distention of the small bowel was found. Through openings in two loops, 5000 cc. of fluid were aspirated. No obstructing lesion was found. An enterostomy was established through the lower opening and the patient made a smooth recovery.

Case 3.—*Recurrent Carcinoma of Left Colon.* Stuart Circle Hospital. This 61-year-old man had partial to complete obstruction for two weeks. He was greatly distended and X-ray examination showed large loops of small bowel. At operation the obstruction

was found in the left colon. The right colon had been resected 2 years before. There was great distention of the transverse colon and small bowel. By suction 3000 cc. of fluid were removed from the small bowel and 2000 cc. from the colon. Tubes were placed in both openings. The patient recovered and the obstructing lesion was resected later.

Case 4.—*Diaphragmatic Hernia: Peritonitis.* This 40-year-old male was operated on at Saint Philip Hospital for strangulated diaphragmatic hernia. The bowel appeared to be viable, but 6 days later he was again explored on account of obstruction. General peritonitis was found from perforation of the small bowel. There was great distention, so the bowel was emptied through 2 openings and enterostomies made. He died 6 days later. It is not unlikely that the bowel would not have perforated if it had been emptied at the first operation.

Case 5.—*Carcinoma of Right Colon.* This 76-year-old male was admitted to the Medical College of Virginia Hospital, having had abdominal pain for 6 weeks. There was complete obstruction following a barium meal. At operation great distention of the cecum and small bowel was found. There was an obstructing napkin-ring lesion in the hepatic flexure. The small bowel was deflated by suction and an enterostomy tube inserted. A cecostomy was then done. The patient recovered from this obstruction but died after ileocolostomy later.

Case 6.—*Band of Adhesions.* This 32-year-old male was admitted to Saint Philip Hospital with gunshot wound of the abdomen. Wounds of small bowel and left colon were repaired. Two weeks later transverse colostomy was done on account of left colon fistula. Two weeks after this, small bowel obstruction developed. At operation the small bowel was obstructed by a band of adhesions and greatly distended. The adhesion was divided, contents of bowel removed by suction and enterostomy established. His recovery progressed satisfactorily except for left femoral thrombophlebitis.

Case 7.—*Abdominal Carcinomatosis.* This 60-year-old man was admitted to Saint Philip Hospital with history of abdominal distention for 3 weeks and no bowel movement for 10 days. Roentgen ray showed great small bowel distention. At operation carcinomatous implants obstructed the small bowel; it was greatly distended. The primary lesion was thought to be in the right colon. The small bowel contents were removed by suction and an enterostomy established. The patient recovered sufficiently to leave the hospital.

Case 8.—*Band of Adhesions.* This 76-year-old woman was admitted to Saint Philip Hospital with acute obstruction of several days duration so close to a moribund state that it was necessary to delay operation for 24 hours. An 8-inch loop of gangrenous ileum bound tightly by a thick adhesion was found, the proximal bowel being greatly distended. The bowel was emptied by suction and an enterostomy established. The gangrenous bowel was resected and end-to-end anastomosis done. Her condition improved steadily to recovery.

Case 9.—*Volvulus of Small Bowel.* This 54-year-old male was admitted to the Medical College of Virginia Hospital with pyloric obstruction from duodenal ulcer. The stomach was resected and a Polya anastomosis done. Two weeks postoperative he developed small bowel obstruction. At operation volvulus of the small bowel around a loop adherent to the omentum was found. There was a recent perforation in the strangulated loop and the bowel above was greatly distended. The perforation was closed, a large amount of fluid and gas was aspirated from the distended bowel, and an enterostomy established. He improved steadily to recovery.

Case 10.—*Strangulated Inguinal Hernia.* This 50-year-old male was admitted to the Medical College of Virginia Hospital with an incarcerated inguinal hernia and great distention of the abdomen. The hernia was reduced but the obstruction was not relieved. The abdomen was explored. A damaged but viable loop of ileum was found and the bowel above was greatly distended. A large amount of fluid was removed by aspiration and an enterostomy established. He made a good recovery.

Case 11.—*Strangulated Inguinal Hernia,* (Dr. C. L. Coleman). This 70-year-old male patient was admitted to St. Philip Hospital with strangulated inguinal hernia. At operation great small bowel distention and gangrenous loop were found. The intestine was emptied by suction, and resection with end-to-end anastomosis performed. This patient had a stormy convalescence but recovered.

Case 12.—*Ileocolic Intussusception,* (Dr. C. L. Coleman). This 65-year-old female patient was admitted to the Saint Philip Hospital with intestinal obstruction. At operation an irreducible ileocecal intussusception based on a malignant tumor was found. The greatly distended small bowel was emptied by suction, the right colon was resected and side-to-side anastomosis done. The patient made an uneventful recovery.

Case 13.—*Ileocolic Intussusception,* (Dr. C. L. Coleman). This 65-year-old female patient was admitted to the Medical College of Virginia Hospital with intestinal obstruction. At operation chronic intussusception was found. The obstruction had recently become complete and the small bowel was greatly distended. The mass was irreducible, so that the right colon was resected, and side-to-side anastomosis done. The patient made an uneventful recovery.

Case 14.—*Volvulus of the Sigmoid Colon,* (Dr. C. L. Coleman). This 50-year-old male patient was admitted to the Medical College of Virginia Hospital. At operation volvulus of the sigmoid colon was found. The ileocecal valve was not competent, so there was great distention of the small bowel as well as the colon. The volvulus was reduced, the small bowel was emptied by aspiration and a rectal tube inserted from the anus to the sigmoid. The patient steadily deteriorated and he died.

SUMMARY OF CASES

These 14 cases all had advanced obstruction with great distention of the small bowel. The obstruction was caused by incarceration in an internal hernia in one case, incarceration and strangulation in inguinal hernia in two cases, strangulation in diaphragmatic hernia one case, strangulation by bands of adhesion in two cases, volvulus in two cases, paralytic ileus in one case, ileocecal intussusception in two cases, and metastatic carcinoma in three cases.

All of them were desperately ill and in four of them resection of the bowel was necessary. Only two patients died and one of these had distention of the colon which could not be relieved by suction, the other had perforation of the involved bowel and general peritonitis.

We believe that restoration of the circulation and of the function of the small bowel by suction at operation was a major factor in the recovery of 12 of 14 patients.

DISCUSSION

It must be understood that we are interested primarily in the question of whether or not to empty the bowel at the time of operation and that restoration of the patient's condition by all the available injections of fluid, electrolytes, and blood is carried out as adequately as time permits. It is frequently difficult to decide exactly how much time can be used for these treatments because a vicious circle is present and the patient is often near the point of irreversible chemical imbalance. The problem is not unlike that of when to operate on a patient shocked by intra-abdominal bleeding. In general it is our belief that efforts at adequate restoration of the patient must be sacrificed to the operation which will remove the cause of the disability.

The remarkable recovery of these patients is strikingly similar to those reported by Holden[6] and Cheever,[1] and is essentially as described by Nicolas Senn.[12]

In all of these cases the bowel was greatly distended and some damage to the wall was present. It was remarkable to see the steady improvement when the bowel had been revitalized by decompression, and the accompanying restoration of circulation in the bowel wall. We feel that there is probably no specific intraluminal toxic substance. There is, however, a varying degree of damage to the wall of the bowel which may be the seat of infection, but the best possible treatment for this is restoration of the circulation.

The condition of the bowel in these cases will not permit delay, so that slow emptying by nasal tubes and by conventional enterostomies is entirely inadequate; the only alternative is to open the bowel at operation and empty it as adequately as possible by aspiration.

It seems to us that the rather complicated apparatus advised by Wangensteen[15] to prevent spillage is not necessary. There is little or no leakage by the use of the ordinary suction apparatus as described above, and in addition, slight soiling is of no consequence as demonstrated by recovery of so many patients with recent perforations of the intestinal tract and by the numerous open anastomoses done today.

After emptying the bowel and completing the necessary operative procedures it is important to keep the bowel empty. This is accomplished by suction on the stomach, by the enterostomy tube, and in some cases by strong purgation.

Of course, in addition, the proper intravenous fluids, electrolytes, blood, and antibiotics must be given after operation.

SUMMARY

1. The effects of great distention of the small bowel are presented.

2. The importance of restoration of the circulation in order to establish function of the bowel is discussed; removal of bowel contents by suction during operation promptly accomplishes this restoration in viable bowel.

3. Continued efforts to prevent distention should be carried out.

4. Fourteen cases are reported.

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FURTHER REDUCTIONS IN THE MORTALITY IN ACUTE APPENDICITIS IN CHILDREN*

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"What we wish to accomplish in the treatment of appendicitis is not to save half of our cases, nor four cases out of five, but all of them."

—Charles McBurney, 1889.

THIS STATEMENT, made in the early days of the surgical treatment of appendicitis, represents the goal toward which surgeons have ever since been striving. Yet for decades it was a discouraging battle, with long casualty lists appearing annually, in which were always included a large proportion of children. Despite better diagnosis, improved hospital facilities and good surgery, the disease and its complications appeared to maintain the upper hand. Finally, however, the break came and about 15 years ago the mortality rates began to fall. Ten years ago, there was a more decided downward trend in the curve, and this further decline in death rate has continued year by year to the present low levels. Although there is not yet room for complacency, there is room for a certain amount of satisfaction. Tribute is due to this Association and more particularly to those of its senior members who throughout the years have been among the leaders of the profession in reducing the death toll from appendicitis. They should derive a sense of satisfaction from the mortality curves which can be shown today.

In order to illustrate specifically, reference is made to Figure 1, showing death rates from appendicitis over the last 20 year period. The three curves represent three sources of statistics, and it is interesting to observe that in general they maintain their relation one to the other, thereby strengthening their validity. The figures for the nation have come from the National Office of Vital Statistics and represent, as might be expected, a slightly higher mortality rate. The second curve comes from statistics derived from the experience of the Industrial Policyholders of the Metropolitan Life Insurance Company. In 1947[2] this Company published under the heading "Appendicitis Mortality Near Vanishing Point," the following optimistic statement: "There is good reason to believe that within the next few years appendicitis will be reduced to a very minor cause of death in our country, and that medical science and public health administration will close another important chapter in their history." Again in 1949[3] with further decreases apparent, this Company noted a 76 per cent reduction over the previous decade in appendicitis mortality, and this was shared by every age period. For the third curve, the experience of the

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City of Baltimore was selected, because the series about to be presented has been largely derived from within its geographical limits. The better experience of this group is in line with better mortality records generally in larger cities. In this city of approximately a million population there were in 1948 exactly 11 deaths from appendicitis recorded with the Statistical Section of the Baltimore City Health Department. This death rate of 1.1 per 100,000 population represents an 86 per cent decrease in this city over the rate of 7.7 for ten

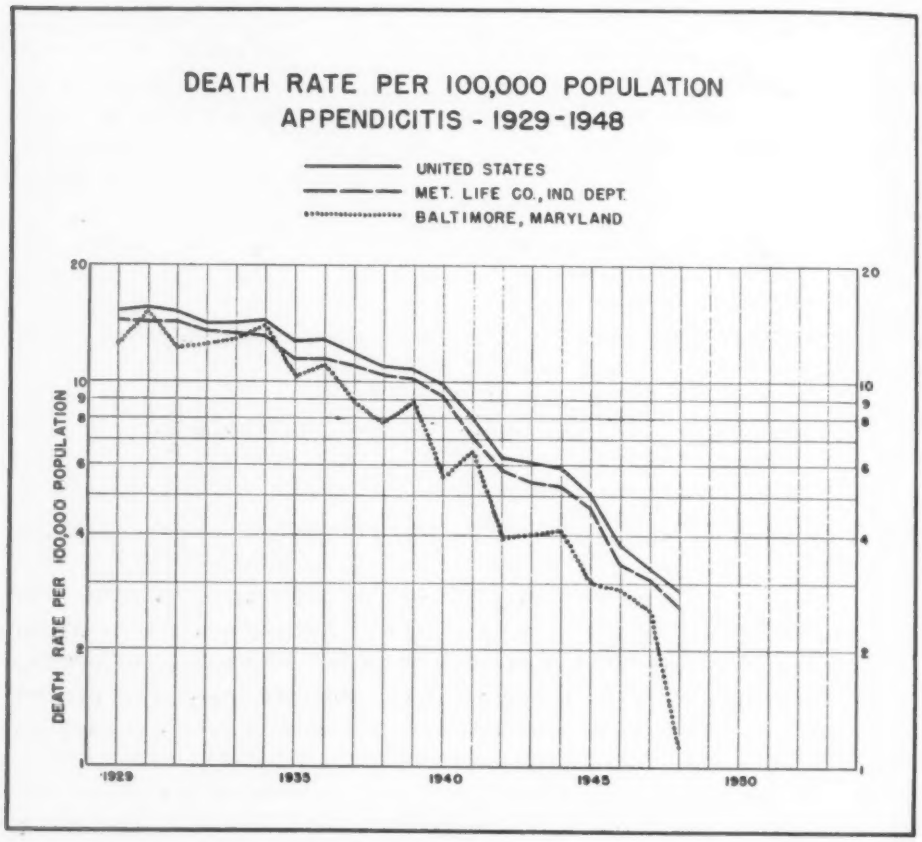

FIG. 1.—Death rate per 100,000 Population Appendicitis—1929-1948.

years ago and a decrease of 91 per cent over the rate of 12.8 for 15 years ago. Only two children under 15 years of age were reported as dying from appendicitis in Baltimore in 1948, and only two in 1947.

There has been an interplay of many factors in producing this lowered mortality. Not the least of these have been the education of the profession in diagnosing and treating appendicitis, and the education of the public in producing an awareness of the disease and in curbing the philocathartic propensities of people generally. Lehman, Hawk and Becker[4] have recently emphasized the fallacy of leaving out of account the drop in mortality of all

surgical diseases, in a comparable period, when commenting on the drop in any one disease. This gives emphasis to the improved surgical care generally, and the many supportive measures with which we are all familiar. The advent of the sulfonamides in 1939 and the introduction of penicillin in 1944 have undoubtedly contributed in a large measure to the further drops in mortality rates after those years.

CLINICAL STUDY

In order to illustrate more particularly what the mortality reduction means, there will be presented briefly some of the findings, the methods of treatment, and the conclusions reached in a study of 823 cases of acute appendicitis in children. These children, 12 years of age and under, have been treated at the Union Memorial Hospital in Baltimore, and represent all of the cases of acute appendicitis with and without perforation in that age group from January 1, 1933, until July 1, 1949, a period of sixteen and one-half years (Table I). A previous study[5] had reported the cases from this hospital prior to 1933.

TABLE I.—*Acute Appendicitis in Children. Data on Union Memorial Hospital Series (January, 1933-July, 1949)*

Total Cases	Patients Operated Upon by Visiting Staff	Patients Operated Upon by Resident Staff
823	535 — 65%	288 — 35%

Number of operators participating: 90.
Deaths in this series: 2. Mortality rate: 0.24 per cent.

These children have been operated upon by both the Visiting Staff and the Resident Staff, the latter including several "generations" of young surgeons in various stages of their training. Actually, 535 cases, or 65 per cent, have been cared for by the Visiting Surgeons, while 288 cases, or 35 per cent, have been operated upon by the Resident Staff. Ninety surgeons have participated, admittedly a large number for this group of patients.

All of the cases included have been confirmed microscopically as acute appendicitis with the exception of those instances where only the drainage of an abscess was carried out and the appendix was not removed. There have been but two fatalities in this entire group, giving a mortality rate of 0.24 per cent.

INCIDENCE

The sex incidence in this series of 823 cases shows the expected predominance of males, who comprised 60 per cent of the total. The disease has been relatively rare, as always, in infants and young children, with a steady increase in incidence as the older ages are reached. Conversely, the relative occurrence of perforation is higher at the younger ages and declines steadily toward the older ages (Table II). Under three years of age, for instance, there were 23 children, of whom 12, or more than half, presented perforated appendices.

During the twelfth year there were 128 cases, of whom 14, or only 11 per cent, were perforated. It is also interesting to note that during the first eight years covered by the study, the incidence of perforation was 17 per cent, while during the second eight years it had fallen to nine per cent.

TABLE II.—*Acute Appendicitis in Children. Relation Between Type of Appendicitis and Age.*

Acute Unruptured		Acute Ruptured	
Age in Years	Number of Cases	Age in Years	Number of Cases
0–2	1	0–2	3
2–4	27	2–4	19
4–6	77	4–6	21
6–8	115	6–8	17
8–10	152	8–10	12
10–13	340	10–13	39
Average age: 8.8		Average age: 7.1 years	

ETIOLOGY AND PATHOLOGY

It is not the purpose of this paper to review the oft-repeated anatomic and physiologic factors which modify the course of the disease in children and lead to earlier perforation. But it is worthwhile to point out the high incidence of fecaliths in children's appendices, as reported by Scott and Ware.[6] They found concretions in 40 per cent of their Children's Hospital series. The occasional occurrence of pin worms (oxyuris vermicularis) may produce symptoms which lead to a clinical diagnosis of appendicitis, yet the appendix may show no microscopic evidence of inflammation.

Pathologically, these cases have all been classified into three groups for analysis (Table III). The first group (712 cases) were those having acutely inflamed appendices without gross evidence of rupture; the second group (82 cases) were those whose appendices had ruptured but who demonstrated only a localized peritonitis or abscess, showing a definite attempt at walling

TABLE III.—*Pathologic Types of Acute Appendicitis and Mortality Rates in 823 Children.*

Type	Number of Cases	Per Cent of Total	Number of Deaths	Mortality Per Cent
Acute, unruptured	712	86	1	0.14
Acute, ruptured with abscess or localized peritonitis	82	10	1	1.21
Acute, ruptured with spreading peritonitis	29	4	0	0.00
Total	823	100	2	0.24

off; and the third group (29 cases) were those whose appendices had ruptured and who gave evidence of a generalized and spreading infection with little attempt at localization. The lack of development of the omentum, especially in the smaller child, has been a factor many times in the failure to wall off the infectious process.

We have been especially interested in observing in these children the condition of the mesenteric lymph nodes where possible, and we have been impressed by the high incidence of visible and palpable adenitis. The nodes lie, as would be expected, in the mesentery of the terminal ileum and cecum, and frequently reach large proportions. As one searches for a Meckel's diverticulum in the uncomplicated case, he has the opportunity to observe the size of the nodes and the extent of the adenitis. The repeated observation that the distribution of the nodes remains essentially the same in the so-called primary mesenteric lymph adenitis, along with the repeated observation that these children recover so quickly after the removal of their "normal" appendix, have convinced this author that the appendix must frequently be the portal of entry for the adenitis. The analogy of the "abdominal tonsil," as the appendix used to be called, with the pharyngeal tonsil, can become still closer if one compares the mesenteric adenitis with the cervical adenitis. The search for a Meckel's diverticulum was successful in seven cases in the group of 712 acute unruptured appendices. In the other groups it would have been bad judgment even to look for a Meckel's diverticulum, and indeed none were recorded as being found. Two of the seven diverticula were not excised because they were of the wide base variety, and the operator in each instance did not believe removal was justified.

TABLE IV.—*Relation Between Type of Appendicitis and the Average Temperature and Leukocyte Count for Each Type.*

	Av. Temperature	Av. W. B. C.
Acute unruptured	100.0°	14,200
Acute ruptured	101.6°	17,926

DIAGNOSIS

Any discussion of diagnosis of this subject should start with the axiomatic principle that, in children, abdominal pain, vomiting and slight fever should always be considered as due to acute appendicitis until proved otherwise. When the diagnosis is difficult, we have been impressed with the value of repeated examinations of the smaller children at reasonably short intervals. It is amazing what a different picture the child may present after a period of one or more hours of observation in or out of the hospital, thereby clarifying many times what may have originally been a confusing situation.

Although this paper has avoided a statistical consideration of all the signs and symptoms in these children, it is interesting to consider briefly the relation of the admission temperature and the leukocyte count to the pathologic process observed (Table IV). The average temperature for the large group of children having appendices without perforation was 100.0 degrees, while the other two groups, one with localized abscess and one with spreading infection, averaged the same temperature, namely 101.6 degrees. The leukocyte counts in children are inclined to be higher than in adults, and this probably explains

the high average count of 14,200 leukocytes in the unruptured cases. The average for the cases of ruptured appendicitis is 17,926 leukocytes, well above the unruptured group. The highest leukocyte count was 41,000 and the lowest 2,000, the latter in an acute unruptured appendix. Our House Officers have been taught to consider an elevated count significant, but not to allow a low count to influence the decision in the face of clear clinical evidence.

Prompt and accurate diagnosis is most essential in the infants and small children where the morality largely occurs. In a previous series[5] I reported an average age of 3.2 years for the children who succumbed, and similar experiences have been reported from many clinics over and over again. The small child gets caught between the Scylla of delayed diagnosis on the one hand and the Charybdis of an inability to localize his infection on the other. The result is all too frequently appendicitis with peritonitis. Therein the morbidity and mortality lie, for the small child has less resistance against peritonitis.

Thought must be given to differential diagnosis in order to exclude some of the more obvious diseases. The abdominal symptoms preceding the onset of the exanthemata, gastroenteritis and mesenteric adenitis have been among the most difficult. One must continually bear in mind the possibility of appendicitis complicating gastro-enteritis when it occurs, either in isolated cases or in epidemic form. We have probably all had the experience of operating upon children or adults in whom the diagnosis had been delayed under these circumstances and have found advanced or complicated appendicitis, much to our own chagrin, or that of our medical colleagues.

TREATMENT

One of the early students of clinical appendicitis in this country, Dr. Alfred Worcester[7] of Waltham, Mass., wrote in 1892: "There is only one logical treatment of the disease, namely the excision of the diseased organ as soon as the diagnosis is made." Through the years that have intervened many have taken issue with this point of view, and for good and sufficient reason. However, in this series of cases extending over sixteen and one-half years this principle has been generally adhered to and prompt operation has been the rule, regardless of the suspected condition of the appendix.

Although there are differences of opinion among surgeons about the management of the complicated cases, there is general agreement that immediate appendectomy should be undertaken for acute unruptured appendicitis. The mortality rates as reported from many clinics for this group approach or actually are zero. In the group being reported, there were 712 cases of uncomplicated appendicitis operated upon with one death, a mortality rate of 0.14 per cent. That death occurred upon the operating table in an infant two years old.

It is with the group of children who present themselves with a mass in the right lower quadrant, and with the other signs and symptoms of perforation, that a controversy as to therapy has existed. Many experienced surgeons

have found that their mortality could be reduced by following a conservative course in adults as well as in children with this picture. Miller and co-workers[8] and later Oberhelman[9] have reported such an experience from the Cook County Hospital, where the mortality rate for children fell to 2.2 per cent. In 1948, Schulz[10] reported his experience with perforated appendix and showed a mortality rate of 3.4 per cent for the non-operative group and 4.7 per cent for the operative group. Our own mortality rate for 111 cases of perforated appendicitis over more than a 16 year period is 0.9 per cent, there being one death from paralytic ileus in a ten-year-old child in 1940. We believe that this result justifies our policy of employing surgery for all stages of appendicitis. It is likely that even the most staunch advocates of conservative treatment for a certain group of these children are finding and will find their group growing smaller and smaller in view of the generally more favorable operative results due to the use of the chemotherapeutic and antibiotic compounds.

Probably one of the most noteworthy advances has been the realization of the necessity of administering adequate preoperative preparation before subjecting the really sick child to operation. This must necessarily be a matter of individualization. The correction of dehydration and ketosis, so common even in children with early appendicitis, requires the administration of dextrose in isotonic sodium chloride solution. Plasma will be indicated in the occasional case of peritonitis, as will be gastric suction where unusual distention or evidence of obstruction has appeared. A number of hours may be required before the desired improvement in appearance, temperature, pulse and degree of distention are achieved. Parenteral sulfonamides and antibiotic therapy can well be initiated at this time in the very ill child.

At operation the anesthetic has usually been open drop ether preceded by a basal anesthetic of one of the barbiturates, or of sodium pentothal in the larger children. We have always favored the McBurney type of incision when the diagnosis seemed reasonably certain, and a number of us employ small incisions, at least to start with, because of their many advantages. In this series, other types of incisions were used initially only 17 times. These incisions included right rectus, lower mid-line, right inguinal and kidney incisions. It was necessary to supplement the McBurney approach by a rectus or lower mid-line incision only four times.

The appearance of the peritoneal fluid, if any, as seen when the peritoneum is opened gives a lead at once as to the presence or absence of real inflammatory changes and their degree. No excess fluid suggests little or minimal disease; a larger amount of clear fluid suggest adenitis as well as appendicitis; and cloudy fluid suggests at least very acute appendicitis. The actual removal of an inflamed, adherent, inaccessible appendix requires a lot of surgical skill. The use of one or more pieces of tape passed beneath the appendix and through the meso-appendix for traction, the suction tip used as an aid in dissection, and the occasional maneuver of "rat-tailing" out the inner coats of a long retrocecal appendix while leaving the serosal and adventitial coverings behind,

have been among the more helpful procedures. Another valued technical point is the realization that the finger gently and deftly used becomes the best available dissector. Ligation of the stump and inversion through one or more purse-strings, when possible, has been the routine procedure. Whether to attempt to remove the appendix or merely to drain the abscess becomes frequently a major decision for the operator. In this series, the original operation was limited to drainage in seven patients. The majority of these were in the younger age group and were very ill at the time of operation. Four of these children returned within a few months for appendectomy. Primary removal of the appendix seems all-important to us unless the situation turns out to be a very critical one. We are inclined to agree with Norris and Brayton[11] who state that with modern antibiotics and chemotherapy, the localizing wall of the abscess need no longer be so meticulously avoided by the operator. The philosophy of drainage for both localized abscess and general peritonitis has not been the same for all the operators. Certainly the younger surgeons have been more generous in their employment of drainage, while some of the senior ones have followed the dictum "when in doubt, don't drain." The writer believes that drainage is not necessary in those contaminations incident to the removal of a gangrenous appendix, but believes that the peritoneal cavity should be drained with one or more soft "cigarette" drains when an abscess or spreading peritonitis has been encountered. Certainly this series has shown complicating abscesses which have developed when drainage has been omitted entirely in suppurative cases, or has been inadequate. Sulfanilamide crystals have been employed locally in most of the perforated cases and occasionally in the non-perforated ones. The value of locally placed sulfonamides has been questioned, especially in view of the increasingly effective antibiotics.

The postoperative care of the simple case need not be made complicated. So frequently today, the younger House Officers are under the impression that intravenous fluids and antibiotics must be used almost routinely. Only the child in this group that develops complications needs special treatment. The children who are doing well may be up as soon as they feel like it and may go home within a few days to a week, depending upon circumstances.

When perforation has occurred, the closest watch is likely to be required if recovery is to be expected. Among other things, the marked lability of a child's acid base equilibrium demands it. In the event that gastric suction is to be employed special attention is directed toward the fluid and electrolyte requirements. If the period of parenteral hydration is prolonged over two or three days, amino acid replacement and transfusion will be needed. An excellent and detailed review of this phase of the postoperative care is given by Norris and Brayton.[11] Following the practice of Dr. John M. T. Finney, Jr., a number of the cases in which distention has been anticipated have received castor oil either immediately after operation per gastric tube while still upon the table, or some hours later when they could swallow. In addition to other means of stimulating peristalsis, this has undoubtedly been effective in many instances in avoiding the development of paralytic ileus. Another

feature which has served to save lives in this series has been the fine co-operation between the pediatricians and surgeons in the care of these children. The interest of the pediatric Resident Staff and the constructive care rendered by them in attending these sick infants have turned the tide on many occasions.

Chemotherapy with divided daily doses of sulfadiazine has been administered to most of the children with complicated appendices since 1939, until two years ago. Since then the drug has been employed less frequently. An analysis of the use of penicillin and streptomycin indicates a lessening of the morbidity and the shortening of hospitalization. The cases are actually too few to draw any conclusions beyond those already known to us from larger series.

Chandler, Long and Ott[12] have shown by an interesting statistical study covering the last 20 years the effect that the sulfonamide and antibiotic agents have had on a wide variety of infections, many of them being reduced to an "irreducible minimum." Today's evidence suggests that aureomycin may prove to be the most effective agent now available in peritonitis due to appendicitis. Before this Association last year, Yeager, Ingram and Holbrook[13] reported favorably upon the value of aureomycin in the treatment of experimental peritonitis in dogs and also cited two successful clinical cases. More recently, Wright and his co-workers[14] have pointed to the extremely wide antibacterial spectrum possessed by aureomycin, and have believed it applicable to the mixed intestinal flora of peritonitis. They have reported upon 52 cases of acute peritonitis, and believe the drug to be of significant value. Aureomycin was administered by them intravenously at first, until the patient was able to take it orally. Five hundred mg. of the drug with 500 cc. of 5 per cent glucose in water were given intravenously twice daily. Later, the oral dosage of 500 mg. was given twice daily and usually continued for an average of 7 to 10 days. In our own community, aureomycin has been used frequently recently in the treatment of peritonitis due to appendicitis, and though statistics are not available, it appears to be rapidly becoming the preferred antibiotic.

Without in any degree detracting from the great value of the new agents, it seems not amiss to call attention to the fact that this series began in 1933, antedating them all by years, and that due credit should be given to the elements of sound surgical judgment and careful surgical technic. Surely these qualities must continue to be fundamental in the treatment of appendicitis.

COMPLICATIONS

Many of these children were desperately ill with suppurative complications. Pelvic or abdominal abscesses occurred as definite entities eight times, and there were a number of instances when an elevation of temperature occurred postoperatively that probably represented abscesses which resolved spontaneously. Of the four serious pelvic abscesses, one drained spontaneously through the incision, one was drained surgically through the rectum and two were drained by the suprapubic approach. Of the four abdominal abscesses,

one drained spontaneously through the wound and three were drained by incision. All recovered. Scott and Ware[6] reported 34 cases of pelvic abscess, and in no instance was operative drainage required. They depended upon a resumption of chemotherapy, if it had been discontinued, and the use of hot rectal irrigations. With this regimen the abscesses appeared either to drain spontaneously or to resorb. Certainly the best practice today with regard to residual pelvic or abdominal abscesses is to follow a conservative course, administer antibiotics, and look for spontaneous drainage or steady resolution of the mass. One seven-year old child underwent multiple operations for the drainage of subphrenic and subhepatic abscesses before recovery. Such areas of suppuration seem less likely to disappear spontaneously, and surgical drainage is indicated. Wound infections occurred 19 times, and illustrate in the main the poor resistance of subcutaneous tissues to implanted infection.

Varying degrees of abdominal distention have occurred, indicating either the presence of frank paralytic ileus or an approach to it. Most of these instances have simply been evidences of the "stormy course" which is to be expected following operation for perforated appendix. Favorable response has followed gastric suction and intravenous fluids. Ileostomy was performed once and that in the ten year old child who succumbed.

There was only one instance of mechanical obstruction due to adhesions requiring re-operation. Though many loops of small intestine were matted together, the release of one obstructed loop gave relief, and recovery promptly ensued.

Pulmonary complications were numerous and included upper respiratory infections (ten times), pneumonia (six times) and atelectasis (four times). A number of other less important complications have been recorded but failed to affect materially the recovery of these patients.

MORTALITY

The first death occurred in 1940 in a ten-year old child who originally had had perforation of the appendix with abscess formation. The infection spread, producing paralytic ileus for which ileostomy was performed and from which the child died seven days later. The postmortem showed peritonitis and paralytic ileus.

The second death occurred in a two-year old child who was being operated upon by the resident surgeon. Respiration and cardiac function ceased before the acutely inflammed appendix had been removed. Death was thought to have been associated with the anesthetic.

Even though these deaths have been few, they represent tragedies. Unfailing interest in this disease, the application of wise surgical judgment and improved surgical skill and the intelligent use of all the accessory measures at our command will be required before we reach that objective which Dr. McBurney was pleased to term as "all of them."

SUMMARY

The mortality rates from acute appendicitis generally have been declining more and more rapidly during the past 15 years. This has been illustrated by a chart, showing curves derived from three different statistical sources to prove the point. One of the curves indicates a drop of 76 per cent in the mortality rate over the last decade.

A clinical study extending over 16 years is presented and includes 823 children under 13 years of age with microscopically proved appendicitis. The younger children had the disease less frequently, yet had it in more complicated form. Eighty-six per cent had, pathologically, acutely inflamed unruptured appendices, while 14 per cent had perforated appendices with either local or spreading peritonitis. It is the younger age group in whom the diagnosis is more difficult and who are unable so often to localize their infection. A mortality rate of 0.24 per cent has been achieved for the entire group, a rate of 0.14 per cent for the simple acute appendicitis cases and a rate of 0.9 per cent for 111 cases of ruptured appendicitis. These results have been achieved through emphasis on the following points:

(a) A policy of prompt operation, regardless of suspected condition of the appendix; (b) adequate preoperative preparation, especially for the ill child; (c) the general employment, with few exceptions, of the McBurney incision; (d) removal of the appendix whenever possible, employing the technic of gentle finger dissection in abscess case; (e) adequate postoperative care with special attention to fluid and electrolyte requirements in the small child; (f) rational use of chemotherapeutic and antibiotic preparations; and (g) co-operation of surgical and pediatric staffs in the diagnosis and care of these children.

While the sulfonamides and antibiotics have contributed greatly, credit must be allowed for the elements of sound judgment and careful technic in this 16-year series. Present practice indicates that residual pelvic and abdominal abscesses can usually be managed conservatively. This is not necessarily true of the subphrenic and subhepatic abscesses. Complicating intestinal distention has generally responded well to conservative measures, while one case only of mechanical obstruction required operation.

Two deaths occurred, one in a child aged ten years from peritonitis and paralytic ileus and one in a child aged two years who died on the operating table before the acutely inflamed appendix could be removed.

Wise judgment, improved skill and the intelligent use of accessory measures at our command will be required to improve further the mortality rates in this disease.

Acknowledgment is made to Dr. Robert H. Clifford of New York for his assistance in compiling much of the data for this paper, and to Dr. William A. Johns and Dr. Seibert C. Pearson for permission to use certain of their statistics, and to Dr. W. Thurber Fales of the Baltimore City Health Department for his help with the mortality chart.

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CONSERVATION OF TISSUE IN REPAIR OF MYELOMENINGOCELE IN THE NEW BORN*

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MYELOMENINGOCELE is one of the more frequent congenital malformations requiring treatment in the new born. Rupture of the sac, actual or imminent, is usually cause for emergency operation. The general surgeon must handle many of these cases, since neurosurgical care often is not available in the emergency.

In only a few instances of overt spina bifida is there a covering of normal skin. In these cases, mostly simple meningoceles, operation may be deferred for an indefinite period. In all others one is faced with the necessity of some sort of repair to secure an adequate skin surface. The simplest operation accomplishing this end may be the desirable procedure.

While many recent authors[1, 6, 7, 8] do not follow the suggestion made by Penfield and Cone in 1932,[9] that the meningeal sac be preserved as an absorbing mechanism for cerebrospinal fluid, it is generally agreed that care should be taken not to destroy functional neural elements. These nerve trunks may arch up along the wall of the sac, some terminating at its apex but others continuing on to leave the sac in various directions. Whether these rootlets arborizing in the sac are largely sensory[6] or contain important motor fibres can be determined only if a stimulating unit[8] is available. Technically these nerve trunks are so intimately a part of the sac that separation is most difficult without damaging the nerves.[2, 5] It seems logical to assume that leaving the sac will cause less destruction of its mural nerve trunks. For this reason in about 50 operations over a 12 year period my tendency has been to remove less and less of the sac.

Separation of the sac from the overlying parchment membrane and especially from a granulating or ulcerated surface is likewise often difficult. Gross, in 1948,[4] demonstrated in large omphaloceles that the amniotic membrane could be buried beneath the surface without complications. Since hearing his paper in January, 1948, I have left a varied amount of the parchment membrane attached to the sac in repairing myelomeningocele.

The thin marginal skin which is useless for closure of the surface may also be preserved, and it provides a firm tissue for anchoring the attached sac to the edges of the defect in the spinal canal. Greene and Wollgast in June, 1949,[3] reviewed the literature on the fate of buried skin, and reported five cases of full thickness skin grafts in hernia repair. Their experimental studies demonstrated that the epithelial elements slowly disappeared and that microscopic cyst formation was of minor degree. A review of microscopic sections of our

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series of spina bifidas reveals that the thin marginal skin contains few hair follicles or glandular structures and the parchment membrane none at all.

This chain of reasoning and observation has led to the concept of repairing myelomeningoceles without removing any tissue whatever.

TECHNIC OF CONSERVATIVE OPERATION

Skin preparation is made with ether, followed by tincture of any appropriate colored dye. Local infiltration of the skin and subcutaneous tissues about the defect with about 10 cc. of .5 per cent procaine suffices for anesthesia. An elliptical incision is made through adequate skin as in the classical operation. The encircling ellipse may be directed transversely or longitudinally, depending on the shape of the defect. Dissection is continued outside of the sac down to the lumbar fascia and the opening in the spinal canal. If the sac has not been ruptured by this stage, it is merely opened in its caudal aspect and the contents inspected. Sutures are then placed to secure the thin margins of the isolated mass to the lumbar fascia. Any excess skin or fat may be trimmed off before this suturing. A few reefing sutures may be taken in the apex of the collapsed sac. Fascial closure is usually not practical. Simple closure of skin and subcutaneous tissue is accomplished by appropriate mobilization of flaps. This closure must be water-tight and without undue tension.

Postoperatively it has seemed desirable to keep the hips elevated rather longer than after excision of the sac with fascial closure, since accumulation of subcutaneous fluid is more likely to occur. Excess fluid may be aspirated as required. A pressure dressing should be maintained until all tendency to fluid collection has ceased. A constipating diet is of value in rectal incontinence. Orthopedic correction of deformities of the lower extremities is begun immediately with traction or casts.

RESULTS

Extreme conservation of tissue has been carried out in repair of nine cases during the past 18 months. In three of these essentially no tissue was removed. There has been no infection of any consequence. There has been no cerebrospinal fluid fistula. Three cases had persistent collection of fluid subcutaneously, but in only one of these were more than two aspirations deemed necessary.

In one recent case with total preservation of all defective tissue, separation of the central portion of the wound occurred after removal of the skin sutures, and the parchment membrane bulged through the area of disruption. In spite of pressure dressings, the recurrent sac, acting as a hydrostatic dilator, enlarged the opening to a diameter of over 2 cm. in the course of a few days. A secondary repair was done. The periphery of the meningomyelocele was found to be firmly attached to the opening in the spinal canal. Already reduction in total mass and increased firmness permitted the application of plicating sutures over the still bulging central portions. This plication was reinforced with leaves of lumbar fascia and the skin closed. In retrospect, the fascial

reinforcement, while more difficult at that time, could have been accomplished at the primary operation. Also a more adequate mobilization of skin flaps to relieve tension should have been done. This two-stage operation resulted in no increase in the relatively minor paralysis of one lower extremity. The anal sphincter remains contractile.

In a second recent case now only five weeks of age a huge defect, 10 by 8 cm., contained prominent elements of conus and nerve roots. There was paralysis of the lower extremities and of the sphincter ani. While any attempt at salvage was debatable, the simplified method of closure was performed without difficulty. Primary healing of the skin has been maintained in spite of considerable accumulation of fluid which has been aspirated at weekly intervals. This infant has developed signs of associated defects which render survival unlikely.

In no case with conservative repair has there been increase in neurologic defect from damage to nerve structures at operation. This is in contrast to several cases in former years where increase in paralysis and loss of sphincter function followed radical excision of the sac.

DISCUSSION

The objection may be raised that this operation is not definitive because the repair is less complete than after reconstruction of a canal with normal tissues after excision of the myelomeningocele. Late secondary repairs have not yet been necessary, but may become desirable in some cases. Probably some sort of fascial repair should be attempted at the primary operation when the mass of tissues left outside the canal is not too bulky. However, this study demonstrated that a fascial roof is not absolutely necessary. Apparently the buried skin and sac stimulate the condensation of a new layer of fibrous tissue beneath the subcutaneous fat.

Another possible objection is the failure to free the conus and cauda for future ascent during longitudinal growth. Actually there is not much evidence that this maneuver prevents later increase in paralysis from traction. It has seemed safer to leave the nerve elements within the sac alone at a primary operation on the new born. Too much dissection of these delicate nerves will do more harm than good.

A long term follow-up in a larger series of cases will be necessary to determine how much tissue should be preserved. The present study only demonstrates that it is possible to leave all components of the defect with a satisfactory immediate result. More time must elapse for a comparison of the later results in these recent cases with about 40 older ones in whom partial or total removal of the sac has been done.

CONCLUSIONS

1. Restoration of an adequate covering of skin and subcutaneous tissue is the main objective in repair of spina bifida in the new born.

2. Nerve fibres arching into the sac may be of present or future functional value. These may be destroyed by radical removal of the sac.

3. The defective covering of granulating surface, parchment membrane, and marginal thinned skin, along with the entire sac and its neural contents, may be preserved and secured beneath the surface without untoward complications.

4. The area of the defect may achieve a satisfactory degree of firmness without the necessity of a secondary fascial roofing-over operation.

5. In selected cases the operation of choice may be plastic closure without removal of any of the defective tissue.

SUMMARY

The desirability is proposed of preserving all possible neural elements in the emergency repair of myelomeningocele in the new born. All components of the defect may be buried beneath the surface and simply covered over with adjacent skin and subcutaneous tissue. Illustrative cases are presented.

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DISCUSSION—DR. ROBERT E. MORAN, Washington, D. C.: I have thoroughly enjoyed hearing the interesting paper presented by Doctor Kredel.

By freeing and advancing a half-crescent rotation flap on each side of the defect, I have been able to obtain adequate closure without embarrassment of blood supply. I have experienced difficulty with skin buried with intact blood supply, as was done in this case; in a considerable number of cases I have had to remove it surgically. This appears to pertain particularly to instances in which the face is the recipient site; sweat glands, sebaceous glands and hair are probably responsible for this complicating feature. On the other hand, dermal grafts buried with no blood supply have given me no trouble. I hope that Doctor Kredel's results are finally as successful as they are now encouraging.

CONGENITAL ATRESIA OF THE SMALL INTESTINE

WITH REPORT OF CASES*

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RECENT REPORTS of congenital atresia of the small intestine are significant because of the decline in mortality during the last ten years as compared to earlier reports. The decline in infant mortality, however, has not kept pace with that reported in adult cases of small bowel obstruction. Several interesting studies relating to congenital atresia of the small intestine have been added to the literature during the past few years. Noteworthy contributions have been offered by O'Neill *et al.*,[1] Potts,[2] Glover and Barry,[3] Miller,[4] Ladd,[5] and Swenson and Ladd.[6]

The history and embryology of atresia and the various etiologic theories, with a résumé of its surgical management, have been elucidated at length by O'Neill *et al.*[1] They reviewed the literature since 1910, when Fockens performed the first successful operation for atresia of the small bowel, found 34 instances of recovery, and added two to make a total of 36 recoveries. There were also 13 with stenosis of the small intestine with recovery—a total of 49 cases. The object of this report is to relate our experiences in the management of 15 patients with congenital atresia of the small intestine. With one exception, these cases have all occurred in the Pediatric Service of the Egleston Memorial Hospital for Children, Atlanta, Ga.

The average practitioner or surgeon sees these cases so infrequently that an accurate diagnosis is often difficult and, hence, proper surgical treatment is unduly delayed. Early diagnosis and early institution of supportive treatment, followed by early operative intervention, are deciding factors in the outcome. Operation should not be delayed longer than is necessary to correct the fluid and electrolyte imbalance and to overcome abdominal distention by the use of the Wangensteen suction. Perforation may occur as early as one day after birth (as shown in Case 15). We believe this delay is a major factor in mortality, second only, perhaps, to the factors of prematurity and associated congenital anomalies. Six of the 15 cases now under discussion showed associated congenital anomalies.

We have been impressed by the smooth convalescence in two cases of late intussusception with complete ileus upon whom operation was delayed until the ileus had been relieved by use of the Miller-Abbott tube with correction of fluid and electrolyte imbalance. In small bowel obstruction in the infant, one is still handicapped in the presence of the well-developed ileus as was the case in adult patients before the Miller-Abbott tube was available. The youngest child in whom we have been able to use the Miller-Abbott tube successfully was

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three months of age. The Levine tube, or a catheter in the stomach, has been of great value in the management of these cases, especially in those seen early and in those with duodenal atresia. It has not been of equal value in cases of low obstruction, seen late, because of the difficulty of intubation in an infant.

SYMPTOMS

The symptoms of small bowel obstruction in the infant vary little from those in the adult; at all ages they depend upon the location of the obstruction. In this series there have been 11 duodenal atresias—seven complete and four partial. Projectile vomiting has been a constant symptom. It usually began on the first day and continued throughout the illness. In all but one patient, the vomitus was bile-stained and frequently contained blood. In ten of these patients, atresia occurred below the ampulla. In one, the vomitus was more mucoid in character and the atresia occurred in the first portion of the duodenum. There was, routinely, upper abdominal distention, often with visible peristalsis, which was reduced by gastric lavage. The gaseous distention was confirmed by roentgenograms.

The patient with atresia above the ampulla had normal meconium stools, while patients with atresias below the ampulla passed stools which were grayish-white and more mucoid in character.

Roentgen ray serial studies of the gastro-intestinal tract in the normal child from birth indicated that gas reached the sigmoid and rectum in seven to ten hours. In atresias of the duodenum and high jejunum, the gas pattern was characteristic, showing a complete absence of gas below the distended point of obstruction if the atresia was complete. When the atresia was not complete, the gas pattern was at times confusing. Malrotation and volvulus may simulate this pattern. In low bowel obstruction, roentgen ray studies with contrast medium, usually barium, are condemned; but in the suspected duodenal atresias, especially those with stenosis, it has been of confirmative value, and in our experience has not given rise to the harmful complications reported by others. Before barium is given, one should be certain that a tracheo-esophageal fistula is not present.

In this series there have been four patients with atresia of the distal portion of the small bowel, one of the jejunum and three of the ileum. In this group, vomiting also has been an early symptom. The vomitus was at first bile-stained, and later more nearly resembled meconium. There has been generalized gaseous distention, confirmed by roentgen ray, frequently associated with dilated veins over the abdomen. In low bowel atresia the roentgen ray studies confirmed the degree of distention, but the site of obstruction was not so definitely located as in duodenal atresia. In this group there was one case of atresia of the ileum with perforation and bile meconium peritonitis. This patient also showed pneumo-peritoneum.

As Ladd[5] has stated so aptly, any infant who vomits persistently after nursing or taking a suitable formula should have a roentgenogram of his abdomen. A diagnosis of obstruction can invariably be confirmed thereby.

As a differential diagnosis between small bowel obstruction and obstruction of the large bowel, Glover[3] has suggested a barium enema. This has not been routine in our series, but it is doubtless valuable, not only for differential diagnosis, but as confirmation of an additional atresia of the colon. The latter is difficult, if not impossible, to confirm at the time of exploration, if the atresia is due to a diaphragm. The percentage of cases of multiple atresia reported by O'Neill *et al.*[1] varies from five to 25 per cent. In our group there was one case of multiple atresia, all located in the ileum.

PREOPERATIVE, OPERATIVE AND POSTOPERATIVE MANAGEMENT

The proper preoperative preparation in these infants cannot be overemphasized. Many are premature. In this series there were three prematures. The infants frequently are admitted markedly dehydrated, and before operation is contemplated, their fluid and electrolyte balance should be well established, with transfusions when indicated. An effort should be made to reduce distention by use of the Wangensteen suction. Proper provision for transfusion during the operation is an absolute necessity. A cannula should be in the infant's veins before the operation is begun.

The anesthetic in this group has been drop ether. The incision has been a liberal right rectus one, with ample exposure for complete exploration of the gastro-intestinal tract. After the nature of the obstruction has been determined, the corrective procedure should be as simple as possible and the operation that best meets this requirement is a short-circuit. We question the wisdom of a too extensive procedure if the obstruction can be relieved by a simple entero-enterostomy. This, of course, does not apply to cases of extrinsic obstruction where there is danger of strangulation. In these, a more extensive procedure often will be necessary. There was only one resection done in this group; the others all had short-circuiting procedures.

In five cases of duodenal atresia we have employed a duodenojejunostomy. In two cases, where this was not feasible, we have done either a posterior or anterior gastrojejunostomy. The anastomosis usually has been done with two rows of 0000 silk. In two cases the distal segment of bowel was unusually small and we used only one layer of interrupted silk in the musculo-serous coat, placing the sutures close together. No clamps were used. Ladd[5] emphasizes the advantages of duodenojejunostomy since, in his experience, gastroenterostomy often causes loss of appetite and failure to gain weight. This has not been a serious handicap in the two cases in whom we have performed gastrojejunostomies.

In patients with low atresias, with one exception (Case 15 with atresia of the ileum with perforation), we have done an entero-enterostomy. In Case 15 the area of atresia was resected, followed by a side-to-side anastomosis. In the infant with multiple atresia, Case 13, a short-circuit entero-enterostomy was performed. There has been some discussion of the ultimate fate of these blind loops left in situ when the short-circuiting procedure is completed, but O'Neill *et al.*[1] report no record of such blind loops perforating, strangulating,

or resulting in cyst formation. To date, approximately two years postoperative, the infant (Case 13) has shown no abdominal symptoms.

Enterostomy has not been employed in this series. It is a universal belief that infants tolerate enterostomy poorly, and it is rarely indicated. O'Neill *et al.*[1] reported only three cases of survival treated by enterostomy alone. Ladd and Gross[7] express belief that if primary ileostomy is done, not more than two or three days should elapse before completing the anastomosis. The consensus is that primary anastomosis is indicated.

The postoperative management in this series has been supervised both by the pediatric and surgical services, with emphasis on careful regulation of fluid and electrolyte balance, blood replacement, early detection of complications, use of the Wangensteen suction to prevent and combat distention, and liberal use of antibiotics, both preventive and corrective. At the present time we prefer penicillin and streptomycin, usually begun when surgery is contemplated. We have found small saline enemas, frequently repeated, advantageous in stimulating peristalsis.

This series includes four cases of congenital atresia of the duodenum and one of the ileum, admitted in such poor condition that operation was believed contraindicated. One patient died of bilateral pneumonia at six days of age; an autopsy was not obtained. One, a Mongolian, showed congenital cardiac anomalies and died on the seventh day of life. The third, a Mongolian, died on the fourteenth day of life and no additional anomalies were demonstrated at autopsy. The fourth infant was premature and died at five days of age. An autopsy revealed multiple anomalies, an atresia of the third portion of the duodenum, a tracheoesophageal fistula, absence of gall bladder, absence of appendix, absence of vagina, bronchial pneumonia, and nephritis. The patient with atresia of the ileum, a two-day-old infant, was admitted to the hospital acutely ill, jaundiced, dehydrated, and comatose. Autopsy revealed atresia of the ileum.

There was one operative death. This occurred in a premature Mongolian with atresia of the second portion of the duodenum. Autopsy showed the anastomosis intact with no evidence of peritonitis. The exact cause of death was undetermined, but was thought to have been due to a fluid and electrolyte imbalance.

There were observed, in addition, one case of fibro-cystic disease of the pancreas with ileus, and one with meconium ileus. These cases gave rise to considerable confusion as to diagnosis, but were not operated upon. In the one with fibro-cystic disease of the pancreas, the diagnosis was confirmed at autopsy and no obstruction was found. Glover[3] reported a similar case that was explored, but no obstruction could be demonstrated and subsequent autopsy confirmed the diagnosis. The case diagnosed meconium ileus was not explored and the patient recovered with conservative management. Neuhauser, as reported by Swenson and Ladd,[6] has stated that a diagnosis can be made in approximately 40 per cent of cases. Assuming that the diagnosis in our case of meconium ileus was correct, it is probably the first survival to be reported.

Hurwitt and Arnheim[8] reported that a survey of the literature up to 1942 failed to disclose any cases that had survived.

The patients not operated upon are mentioned again to emphasize the desirability of close cooperation between the pediatrician, the roentgenologist and the surgeon, which we believe has definitely contributed to the lower mortality reported in this series. There were five non-operative deaths and one operative death—a total mortality of 40 per cent, with an operative mortality of 10 per cent.

SUMMARY

Fifteen cases of congenital atresia of the small intestine have been reported in this series. Five patients were admitted in such poor condition that surgery was not believed indicated; all five died. Ten were operated upon, with one death, an operative mortality of 10 per cent. Four of the five patients who died without operation showed associated congenital anomalies. Close co-operation of the pediatric and surgical services is offered as an explanation of the lower mortality in our series.

CASE REPORTS

Case 1.—S. A. N., a 3-day-old infant girl, was admitted with a diagnosis of intestinal obstruction. The baby did not nurse well and had vomited everything taken by mouth. All vomitus contained bile and occasional small amounts of old blood. A thin barium meal and roentgen ray studies revealed a complete obstruction in the second portion of the duodenum and no gas pattern beyond this point. The baby died on the sixth day of life with bilateral pneumonia. There was no autopsy.

Case 2.—R. V. R., a 48-hour-old Mongolian infant boy, was admitted with complaint of persistent vomiting and short periods of apnea. Roentgen ray studies with thin barium revealed a tremendously dilated stomach and first portion of duodenum, with no evidence of gas below this point. The baby became quite cyanotic and remained so, despite the administration of oxygen. He died on the seventh day of life. Autopsy revealed a diaphragm septum in the first portion of the duodenum. There was a small valve-like flap in the center of the septum through which a small probe barely could be passed. Congenital cardiac anomalies also were present.

Case 3.—T. G. D., a 3-day-old Mongolian infant boy, was admitted with a history of vomiting since birth. The baby had vomited everything taken by mouth and occasionally there was a small amount of blood noted in the vomitus. It was reported that the infant had had no bowel movement. A mild jaundice was present. Roentgen ray studies following insufflation under fluoroscopic observation revealed a markedly dilated stomach and first portion of duodenum which was consistent with the diagnosis of atresia of first portion of duodenum. The baby died on fourteenth day of life.

Case 4.—C. J. B., a one-day-old premature infant girl, was admitted because of vomiting and imperforate anus. Roentgen rays revealed a large dilated stomach and duodenum, but no evidence of intestinal gas. A diagnosis of multiple congenital anomalies, including tracheo-esophageal fistula, was made. The infant died on the fifth day of life. Autopsy revealed atresia of the third portion of duodenum, tracheo-esophageal fistula, absence of gallbladder, absence of sigmoid and rectum, absence of appendix, absence of vaginal introitus, bronchial pneumonia and secondary nephritis.

Case 5.—M. A. W., a 16-day-old infant girl, was admitted with a history of vomiting since the fourth day of life. The baby retained one to two feedings each day and continued to have bowel movements. Roentgen ray examination revealed a large duodenal and gastric residue 4 hours following barium meal. At 24 hours there was still a small amount of barium present in the stomach and duodenum. At operation the stomach and duodenum were moderately dilated and the duodenum was found to be obstructed at a point where it passed through the fetal mesentery of the ascending colon. A retrocolic duodenojejunostomy was performed. One month after operation the baby was operated on again and an obstructing adhesive band was released, following which the baby did very well and was discharged from the hospital in good condition.

Case 6.—M. C. B., a 7-day-old infant girl, was admitted with a history of jaundice and vomiting since birth. Aspiration of stomach revealed the presence of bile. Roentgen ray examination revealed a complete obstruction to thin barium at second portion of duodenum. There was a small gas pattern distal to the duodenum. At operation the stomach and first portion of duodenum were found to be markedly dilated and an obstruction was apparent in the third portion of duodenum. A retrocolic duodenojejunostomy was done. The baby made an uneventful recovery.

Case 7.—B. M. B., a 6-day-old infant boy, was admitted with a history of vomiting since the first day of life. Roentgen ray studies revealed a large dilated esophagus, stomach and proximal duodenum. No gas pattern was apparent below the point of obstruction. At operation the duodenum was markedly dilated, and an obstruction was apparent in the third portion. The remainder of the gastro-intestinal tract was collapsed markedly, and no other abnormalities were evident. A retrocolic duodenojejunostomy was performed. The baby made an uneventful recovery.

Case 8.—M. R. L., a 6-day-old infant boy, was admitted with a history of vomiting since birth. There had been small bowel movements of meconium for 3 days. Roentgen ray examination revealed complete duodenal obstruction in the second portion of duodenum. There was no evidence of gas distal to the obstruction. At operation, stomach and proximal duodenum were dilated and obstruction was present in the second portion of the duodenum. The remaining gastro-intestinal tract, including the colon, was collapsed and no additional abnormalities were evident. An anticolic duodenojejunostomy was performed. The baby made an uneventful recovery.

Case 9.—M. P. C., a 6-day-old infant girl, was admitted with a history of vomiting since birth. The child had never had a bowel movement. Roentgenograms had been brought into the hospital with the patient. Barium, which had been given 24 hours previously, was still pooled in the stomach and first and second portions of duodenum. There was no barium or gas beyond the obstruction. At operation, the stomach and first portion of duodenum appeared to be slightly dilated, and obstruction was apparent in the second portion of the duodenum. Remainder of the gastro-intestinal tract was completely collapsed. An anticolic gastrojejunostomy was performed. The infant made an uneventful recovery.

Case 10.—M. G. A., a 6-hour-old premature infant girl, admitted with a history of cyanosis since birth. Following admission to the hospital the baby vomited everything taken by mouth despite changes in formula and antispasmodics. Roentgen ray studies revealed an almost complete obstruction at the pylorus. There was about 25 per cent gastric residue of barium at 52 hours (Fig. 1). At operation the stomach was found to be dilated markedly and a stenosis was present in the first portion of the duodenum. An anticolic gastrojejunostomy was performed. This baby recovered and was discharged in good condition.

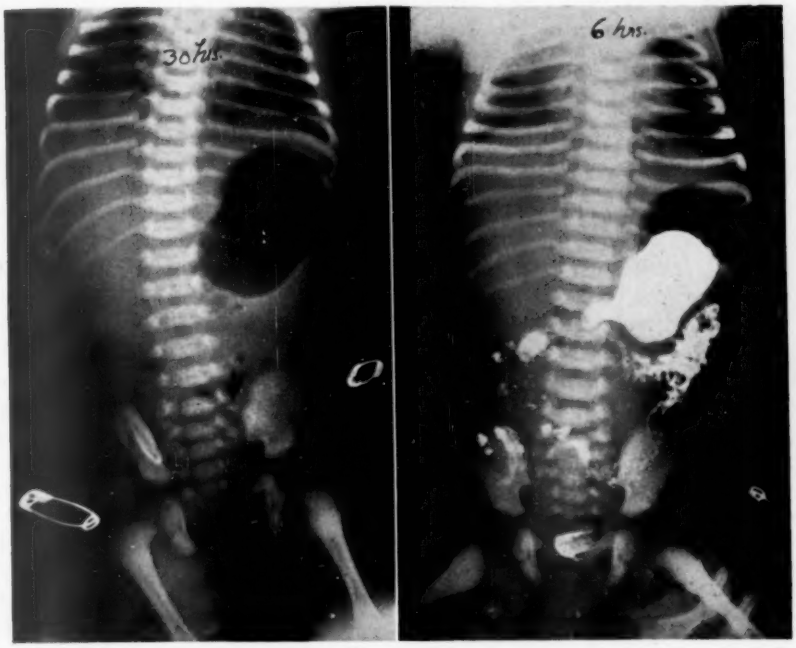

FIG. 1.—(Case 10) Gas pattern of duodenal stenosis confirmed by roentgen ray with thin barium.

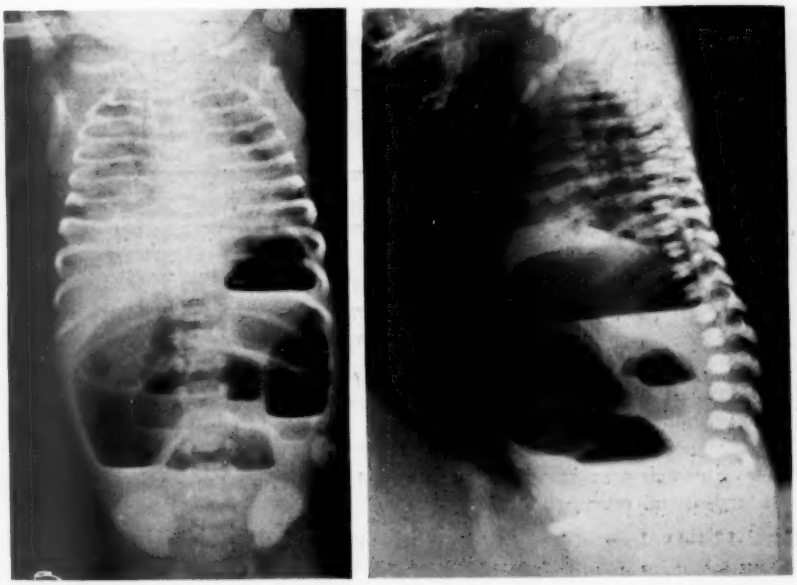

FIG. 2.—(Case 13) Gas pattern of complete atresia of ileum.

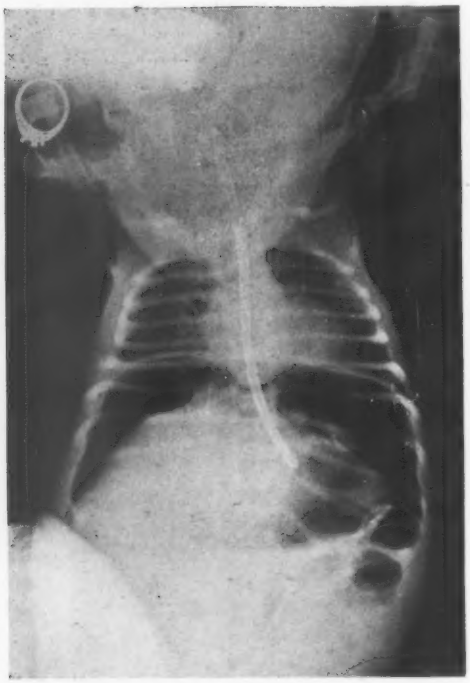

FIG. 3.—(Case 15) Atresia of the ileum with perforation and pneumo-peritoneum.

Case 11.—J. J., a 3-day-old Mongolian, one month premature, male infant, was admitted with a history of persistent vomiting. Roentgen ray studies revealed a large distended stomach and first and second portions of duodenum. There was no gas in the remaining portion of the gastrointestinal tract. At operation the stomach and first portion of the duodenum were found to be dilated markedly and obstruction was apparent in the second portion of duodenum. An anticolic duodenojejunostomy was performed. The baby died on the fourth postoperative day. Autopsy revealed minimal atelectasis. The anastomosis was intact and functioning well. There were no autopsy conclusions regarding death.

Case 12.—C. R., a 2-day-old infant boy, was admitted with a history of vomiting since birth. The child had had several stools, all of which were described as acholic. The baby was jaundiced, acutely ill, dehydrated and semicomatose. The baby died 6 hours after admission to the hospital. Autopsy revealed atresia

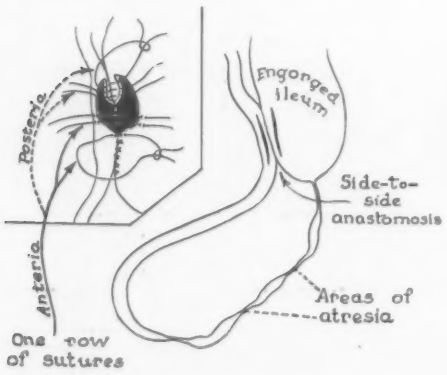

FIG. 4

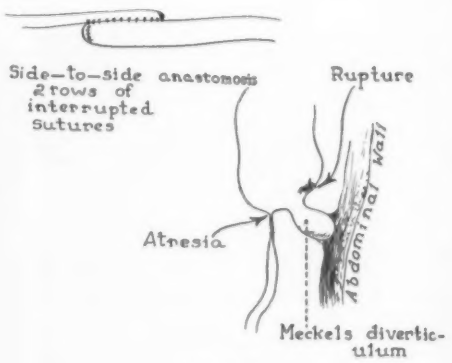

FIG. 5

FIG. 4.—(Case 13) Multiple atresias of the ileum; technic of ileo-ileostomy.
FIG. 5.—(Case 15) Atresia of the ileum with perforation; resection with side-to-side anastomosis.

of ileum. Stomach was small and contracted; however, the upper small bowel was quite distended and cyanosed.

Case 13.—D. J., a 4-day-old infant boy, was admitted with a history of vomiting everything taken by mouth since birth. There had been also some respiratory difficulty and cyanosis. The abdomen was distended markedly, and dilated loops of bowel were visible. Roentgen ray studies revealed marked dilatation of the small bowel. The colon contained a small amount of gas, but was not distended (Fig. 2). A roentgenographic

diagnosis of questionable bilateral cystic disease of the lungs was also made. At operation three areas of atresia were found in mid-ileum. A side-to-side ileo-ileostomy was performed (Fig. 4). Following operation the abdomen remained moderately distended and the baby vomited at frequent intervals. He finally recovered, however, and was discharged in good condition.

CASE NO. & SEX	AGE IN DAYS	TYPE OF OBSTRUCTION	LOCATION OF OBSTRUCTION	AGE AT OPERATION (DAYS)	SURGERY	OTHER ANOMOLIES	RESULTS
1. F. B.A.H.	3	ATRESIA	DUODENUM 2ND. PORTION				DIED 6TH DAY PNEUMONIA
2. M. R.V.R.	2	STENOSIS	DUODENUM 1ST. PORTION			1. MONGOLISM 2. CARDIAC ANOMOLIES	DIED 7TH DAY
3. M. T.S.D.	3	ATRESIA	DUODENUM 1ST. PORTION			1. MONGOLISM	DIED 14TH DAY
4. F. S.A.B.	1	ATRESIA	DUODENUM 3RD. PORTION			1. PREMATURE 2. TRACHEO-ESOPHOGEAL FISTULA 3. ABSENCE SIGMOID AND RECTUM	DIED 5TH DAY
5. F. M.A.W.	16	STENOSIS	DUODENUM 3RD. PORTION	17	DUODENOJEJUNOSTOMY RETROCOLIC	1. MALROTATION	SATISFACTORY
6. F. M.C.B.	7	STENOSIS	DUODENUM 3RD. PORTION	9	DUODENOJEJUNOSTOMY RETROCOLIC		SATISFACTORY
7. M. B.M.B	6	ATRESIA	DUODENUM 3RD. PORTION	7	DUODENOJEJUNOSTOMY RETROCOLIC		SATISFACTORY
8. M. M.R.L.	6	ATRESIA	DUODENUM 2ND. PORTION	7	DUODENOJEJUNOSTOMY ANTICOLIC		SATISFACTORY
9. F. M.P.C.	6	ATRESIA	DUODENUM 2ND. PORTION	7	GASTROJEJUNOSTOMY ANTICOLIC		SATISFACTORY
10. F. M.G.A.	1	STENOSIS	DUODENUM 1ST. PORTION	7	GASTROJEJUNOSTOMY ANTICOLIC	1.PREMATURE	SATISFACTORY
11. M. J.J.	3	ATRESIA	DUODENUM 2ND. PORTION	3	DUODENOJEJUNOSTOMY ANTICOLIC	1.MONGOLISM 2.PREMATURE	DIED 9TH. P.O. DAY
12. M. O.R.	2	ATRESIA	DISTAL ILEUM				DIED 2ND. DAY
13. M. D.J.	4	ATRESIA	ILEUM	9	ILEO-ILEOSTOMY SIDE TO SIDE	1.MULTIPLE AREAS OF ATRESIA 2.CYSTIC DISEASE OF LUNGS ?	SATISFACTORY
14. F. L.A.W.	3	ATRESIA	JEJUNUM	4	JEJUNO-JEJUNOSTOMY SIDE TO SIDE		SATISFACTORY
15. M. S.S.	1	1.ATRESIA 2.VOLVULUS	ILEUM	3	RESECTION OF SEGMENT OF ILEUM & SIDE TO SIDE ANASTOMOSIS	OMPHALOMES-ENTERIC DUCT REMNANT	SATISFACTORY

FIG. 6

Case 14.—L. A. W., a 3-day-old infant girl, was admitted with a history of vomiting since birth. There had been no bowel movement, although one enema had resulted in a hard, gray stool. Roentgen ray examination revealed a large dilated stomach and duodenum. At operation the stomach, duodenum and proximal jejunum for a distance of 10 cm. were found to be enormously dilated. There was an atresia at this point in the jejunum which was approximately 10 cm. in length. This segment of jejunum appeared to be only a thin thread. A side-to-side jejunojejunostomy was performed.

The remaining portion of the gastro-intestinal tract appeared to be normal. The baby continued to vomit for several days, but recovered and was discharged from the hospital in good condition.

Case 15.—S. S. was an infant boy who persistently had vomited everything taken by mouth since birth. On the second day of life the abdomen became distended and rigid. Roentgenograms on the third day revealed a large amount of free air and fluid in the peritoneal cavity (Fig. 3). At operation there was a large amount of meconium present in the abdominal cavity, resulting in a rather marked meconium and bile peritonitis. A perforation was present just proximal to an atretic area of the ileum. Due to an incomplete obliteration of the omphalo-mesenteric duct, there was also present a volvulus involving this segment of the ileum. The portion of ileum involving the

SUMMARY

LOCATION OF OBSTRUCTION	NO. CASES	TOTAL DEATHS	OPERATIVE DEATHS	TOTAL MORTALITY	OPERATIVE MORTALITY
DUODENUM	11	5	1	45.4%	14.3%
JEJUNUM	1	0	0	0	0
ILEUM	3	1	0	33.3%	0
TOTAL	15	6	1	40%	10%

FIG. 7

atresia was resected, the proximal and distal ends closed, and a side-to-side anastomosis performed (Fig. 5). The baby made a surprisingly uneventful recovery, and 6 months later was in good health.

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THE SURGICAL SIGNIFICANCE OF ANOMALIES OF INTESTINAL ROTATION*

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A CLEAR UNDERSTANDING of the normal process of intestinal rotation and of its anomalies is essential to the surgeon. Knowledge of the normal process of rotation and fixation is utilized whenever the colon is mobilized for resection. Unless the surgeon also understands the various possibilities of abnormalities of rotation he may be completely bewildered by the unusual disposition of the viscera when confronted by one of these anomalies and may have to close the abdomen without correcting the condition present.

Anomalies of intestinal rotation are not uncommon. Our interest in them was first aroused in 1934 by an experience with two cases, within a month, of volvulus of the entire mesentery secondary to anomalies of rotation and fixation.[8] In the past year we have seen four infants with volvulus of the entire midgut and one with a huge omphalocele. Because these conditions are still poorly understood by many surgeons it seemed wise to record again the normal steps in the process of intestinal rotation and fixation and to describe abnormalities in each stage as we have encountered them at the Duke Hospital.

Most of our knowledge of intestinal rotation dates from the work of Mall[16] in 1898 and Frazer and Robbins[7] in 1915. Dott[5] was the first to apply this knowledge to practical surgical problems in a masterful article published in 1923. Since then anatomists, pediatricians, gastro-enterologists, roentgenologists, and surgeons have recorded experiences which indicate that anomalies of intestinal rotation are more common than originally believed. When we reviewed the world literature in 1934 105 cases of intestinal obstruction from anomalies of rotation were collected. The literature since then includes at least an equal number of cases, 19 being reported by McIntosh and Donovan.[17]

EMBRYOLOGY

The intestinal tract of the early embryo is a straight structure suspended in the sagittal plane on a common dorsal mesentery. The process by which this primitive position is converted to that seen at birth is called intestinal rotation.

Embryologists divide the primitive intestinal tract into foregut (mouth to duodenojejunal junction), midgut (duodenojejunal junction to midtransverse colon), and hindgut (midtransverse colon to anus). The midgut loop is the one primarily concerned in intestinal obstruction and is the only one under discussion in this presentation. Frazer and Robbins divide the process of

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rotation of this midgut loop into three stages, each of which will be described along with its anomalies.

FIRST STAGE OF MIDGUT ROTATION

Embryology. The primitive intestinal tract is a straight structure suspended on a dorsal mesentery in the sagittal plane (Fig. 1A). As it elongates, the midgut loop bulges through the umbilical orifice into the primitive umbilical cord as a temporary physiologic umbilical herniation. The first stage of intestinal rotation is a contraclockwise rotation of 90° of this intra-umbilical loop from the sagittal (Fig. 1A) to the horizontal plane (Fig. 1B). It occurs at about the eighth week of intra-uterine life.

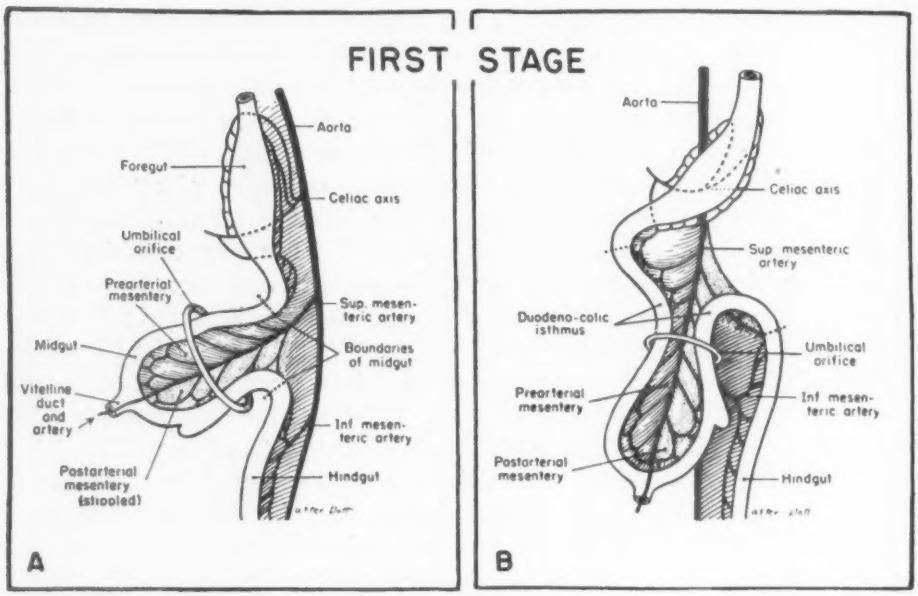

FIG. 1.—First stage of intestinal rotation. (A) Diagrammatic representation of the intestinal tract in the early embryo. The entire intestinal tract hangs suspended in the sagittal plane on a common dorsal mesentery. As the bowel develops the midgut loop bulges into the umbilical stalk as a temporary physiologic umbilical herniation. At about the eighth week of intra-uterine life a 90° contraclockwise rotation of this intra-umbilical, midgut loop from the sagittal plane (A) to the horizontal plane (B) occurs.

Omphalocele. Failure of rotation beyond the first stage and retention of the midgut loop in the umbilical stalk at the time of birth is called omphalocele, amniotic hernia, or exomphalos (Fig. 2). In this condition herniation of intestine and sometimes also of liver and spleen into the umbilical cord is present at birth, the hernial covering being the thin translucent umbilical cord structure.

This anomaly was described by Cullen[3] in his monumental work on the umbilicus as an amniotic hernia. Ladd and Gross[12] refer to it as an ompha-

locele and Dott[5] as exomphalos. It may exist in all degrees of severity. Small herniations may only contain a single loop of bowel. Others, as ours (Fig. 2), may contain the entire intestinal tract, liver, spleen, and pancreas, and be larger than the child's abdominal cavity.

The jellylike cord structure covering such a herniation is delicate and liable to rupture within the first few hours of life. To prevent evisceration it is necessary to close the defect as promptly as possible after delivery. In small herniations peritoneum, fascia, and skin closures can be obtained. In larger herniations it is possible only to close the skin. Hollenberg[10] reports

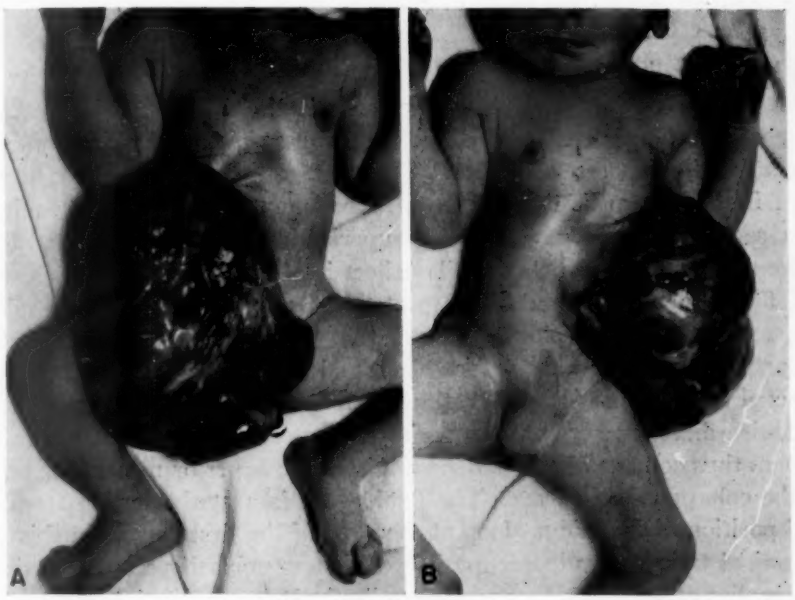

FIG. 2.—Omphalocele (Case 1). Rotation has been arrested in the first stage. The child was born with abdominal viscera retained in the umbilical stalk. Coverings of the hernia are the delicate, jellylike, translucent cord structure. The tie on the umbilical cord is seen at the lower margin of the herniation (A). B shows the transition between skin and cord structure covering the hernia.

four successful cases in which skin was closed over the viscera after the covering membrane had been removed.

Gross[9] more recently has described a technic for the treatment of large omphaloceles in which the amniotic membrane is not removed. Skin mobilized from about the umbilical orifice is utilized to cover the herniated viscera without first removing the covering membrane. After six to 12 months the fascia and muscles are closed over the defect. This technic eliminates evisceration of bowel at operation, and since the viscera are not replaced into the peritoneal cavity there is minimum rise in intra-abdominal pressure, with crowding of the viscera against the diaphragm.

Case 1.—*Omphalocele.* B. B., No. C-46124, a male Negro infant, was seen 12 hours after delivery with a huge omphalocele (Fig. 2). At operation 2 hours later skin over the entire anterior and both lateral portions of the torso was mobilized and used to cover the herniation, no effort being made to remove the translucent cord structure covering the herniation (Gross' technic). The child died 2 hours later. At autopsy the herniation contained liver, pancreas, spleen, and midgut. The lungs were atelectatic. The heart was not completely rotated. The foramen ovale and the ductus arteriosus were patent.

SECOND STAGE OF MIDGUT ROTATION

Embryology. This is the stage when reduction and major rotation of the bowel occurs and is the most important stage of intestinal rotation. The midgut loop returns to the peritoneal cavity from its temporary position in the umbilical herniation and at the same time rotates an additional 180° in a contraclockwise direction about the mesenteric root as a pedicle (Fig. 3). This stage occurs quickly, at about the tenth week of intra-uterine life, and in none of the models studied by Mall or by Frazier and Robbins was the gut found in its process of return. The proximal limb of the pre-arterial segment is thought to be reduced first, its coils entering the abdomen in an orderly sequence, passing under the superior mesenteric vessels and the mesentery (Fig. 3B). As these coils collect in the left side of the abdomen they deflect the hindgut and its mesentery to the left so that the splenic flexure and descending colon are carried into their normal position. The cecum and adjacent colon are reduced last, and as the colon straightens out it is deflected to the right (Fig. 3C), thus completing a 180° rotation in a contraclockwise direction about the superior mesenteric artery as an axis. The duodenum thus comes to lie under the origin of the superior mesenteric artery while the colon passes in front. The final result is a 270° rotation from the sagittal position of the midgut loop at the start of the first stage. In this way the intestinal tract comes to occupy its position as normally seen at birth.

Anomalies of the second stage of rotation include nonrotation, volvulus of the midgut, malrotation, internal hernia, and reversed rotation.

Nonrotation. In nonrotation the midgut loop is returned to the peritoneal cavity from the temporary umbilical herniation without having rotated beyond the horizontal plane it occupied at the end of the first stage (Fig. 1B). The duodenum descends on the right of the superior mesenteric artery (Fig. 4A). The small intestine is entirely in the right side of the abdomen and the colon is on the left. The cecum is in the left lower quadrant. The terminal ileum crosses the midline to enter the cecum from the right. From this point the ascending colon passes upward on the left of the midline to a point behind the greater curvature of the stomach. Between this point and the splenic flexure is a narrow U-shaped loop of transverse colon.

Nonrotation may exist without symptom. Roentgenologists frequently see it as an incidental finding during examination of the barium filled colon and refer to it as "left-sided colon." Its major surgical significance occurs when diseases of the appendix, cecum or ascending colon occur in an unusual position.

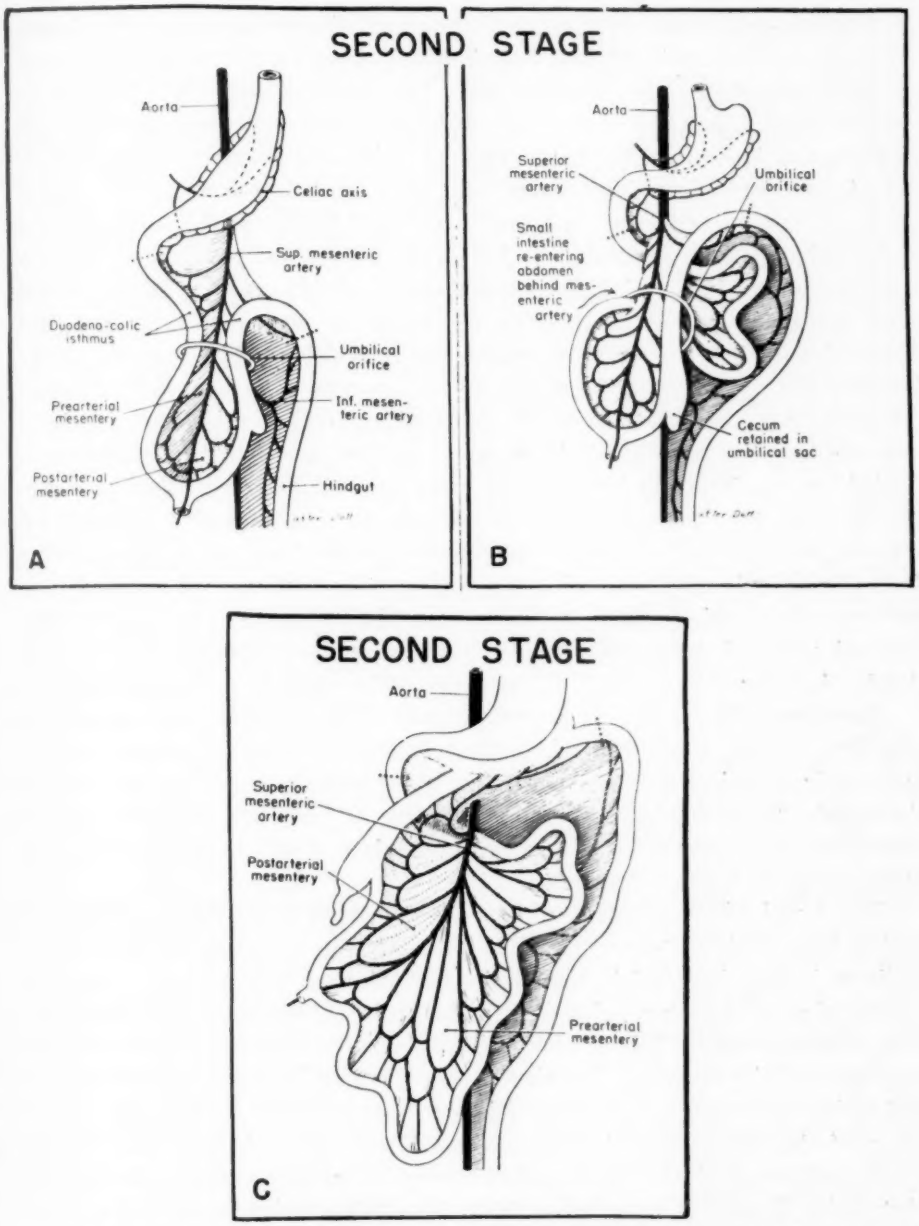

FIG. 3.—Second stage of intestinal rotation. The midgut loop which occupies a position in the umbilical stalk at the beginning of the second stage (A) is reduced back into the peritoneal cavity (B) and in so doing rotates 180° in a contraclockwise direction about the superior mesenteric artery as an axis. The proximal loop of midgut re-enters the peritoneal cavity under the mesenteric root first (B). An orderly reduction of the remainder of the midgut loop then occurs, the cecum and ascending colon being reduced last. As the cecum and ascending colon straighten out they are deflected to the right upper quadrant, completing the 180° contraclockwise rotation of the midgut loop about the mesenteric root. In this way the duodenum is thrown under the superior mesenteric artery and the colon passes in front of it. This stage of rotation occurs quickly at about the tenth week of intra-uterine life.

Volvulus of Midgut. If nonrotation occurs without secondary fixation of the mesentery the entire midgut loop hangs free from a narrow pedicle at the origin of the superior mesenteric artery. The origin of the midgut loop at the duodenojejunal junction and its termination at the midtransverse colon are in close proximity to each other (Fig. 3A). Such a position predisposes to volvulus of the entire midgut loop (Fig. 5A). In other situations of failure of complete rotation where the cecum is arrested in the right upper quadrant and fixation of the mesenteric root fails to occur, a narrow mesenteric pedicle from cecum to duodenojejunal junction may permit volvulus of the entire small intestine. In our original report[8] about 25 per cent of the recorded cases of volvulus were of the entire small intestine while the remainder included the right half of the colon.

Symptoms from volvulus of the entire midgut will depend upon the tightness of the twist, whether it is recurrent or not, and whether it is accompanied by vascular occlusion. In most cases the volvulus occurs in intra-uterine life or shortly after birth, and the twist is not tight enough to give symptoms of vascular occlusion or of obstruction to the loop of bowel emerging from the volvulus. The usual symptoms are those of partial, complete, or recurrent duodenal obstruction dating from birth (Case 2, 3 and 4). In our original collected series[8] 70 per cent of the cases gave symptoms of chronic duodenal obstruction and half of the entire series were in newborn infants.

Sometimes the volvulus may occur acutely without previous symptoms and give rise to symptoms of acute intestinal obstruction (Case 5). Symptoms of mesenteric vascular occlusion with circulatory collapse may also accompany the attack. Blood in the vomitus and passage of blood per rectum has been recorded.[8] Our Case 5 had three bloody stools and was thought to have an intussusception before operation.

The usual picture is that of partial or complete duodenal obstruction dating from birth (Cases 2, 3 and 4). First feedings are usually taken normally and meconium and in some cases normal stools are passed. Vomiting, constipation, and abdominal pain usually begin on the third or fourth day. The vomitus contains bile. This finding rules out congenital pyloric stenosis as a cause of the vomiting. Vomitus is often projectile. Epigastric distention and visible waves of gastric peristalsis are often present. A plain roentgenogram of the abdomen will show a dilated stomach and duodenum with a small amount of air in the intestine, or not, depending upon whether the obstruction is partial or complete (Fig. 6). It is unwise to put barium into the stomach. If the roentgenologist is able to demonstrate that the entire duodenum is on the right side of the vertebral column or that the colon is entirely on the left an anomaly of rotation can be suspected. Usually it is impossible to determine before operation whether the duodenal obstruction is caused by atresia, stenosis, extrinsic bands, or a volvulus of the midgut.

Operative treatment offers the only hope of cure for patients with volvulus of the midgut. In those with symptoms of intestinal obstruction or vascular occlusion immediate operation is imperative (Case 5). In the larger group,

with symptoms of chronic duodenal obstruction, a more leisurely preparation for operation may be pursued. At operation the volvulus of the entire mesentery may be easily overlooked if adequate exploration is not done. The condition may be suspected if the right half of the colon is not found in its normal position or if on palpation a firm cord representing the twisted root of the mesentery can be felt at the site of origin of the superior mesenteric artery. Complete evisceration of all of the intestine is advised as the quickest method of determining the true nature of the condition present. Detorsion of the volvulus is easily accomplished. All of our patients had adhesions attaching bowel and mesentery of the entering and emerging loops of intestine which had been involved in the volvulus (Fig. 5B). These adhesions must be divided as in freeing two adherent leaves of a book. Adhesive bands may also extend from the colon across the duodenum to the posterior abdominal wall. They must be divided, as well as the adhesions uniting entering and emerging loops of bowel, in order that the duodenum be completely freed and separated widely from the colon. If these adhesions are not freed recurrence of the volvulus may occur.

Chronic venous obstruction which has accompanied the volvulus may cause enormous distention of the mesenteric veins (Fig. 5C). Care must be taken not to damage these veins when adherent leaves of mesentery are separated. Lymph nodes in the mesentery are usually much enlarged by the lymphatic obstruction which accompanied the volvulus. In our Case 5 dilated lymphatics could be seen in the mesentery and there was a little chylous free fluid in the peritoneal cavity.

After being freed the bowel is returned to the abdomen in a state of nonrotation, with the duodenum descending on the right of the vertebral column and the colon ascending on the left (Fig. 5C). Entering and emerging loops of bowel involved in the volvulus which were previously adherent are separated as widely as possible when the bowel is replaced. No effort to fix the bowel has been attempted in any of our cases and none of our patients have had recurrence of their volvulus. Recurrences have been reported in cases where adhesions between loops of bowel in the volvulus or adhesive bands crossing the duodenum were not freed.[2, 13, 20] Wangensteen[21] has reported a method of fixation of the bowel in a position of normal rotation. Some effort to fix the nonrotated cecum and ascending colon along the left lumbar gutter would seem to be a simpler method of preventing recurrence of the volvulus, if any fixation is necessary.

Case 2.—*Volvulus of Midgut.* W. D., No. C-48290, was an 8-week-old Negro male with history of vomiting of bile-stained material since the age of 6 days. Bowel movements had been normal. On examination he was dehydrated and undernourished. Roentgen rays showed a stomach hugely distended with gas (Fig. 6A). At operation a 360° clockwise twist of midgut loop was encountered (Fig. 5A). After detorsion and separating of adherent entering and emerging loops of bowel, the bowel was replaced in the abdomen so that the small intestine was in the right half and colon in the left

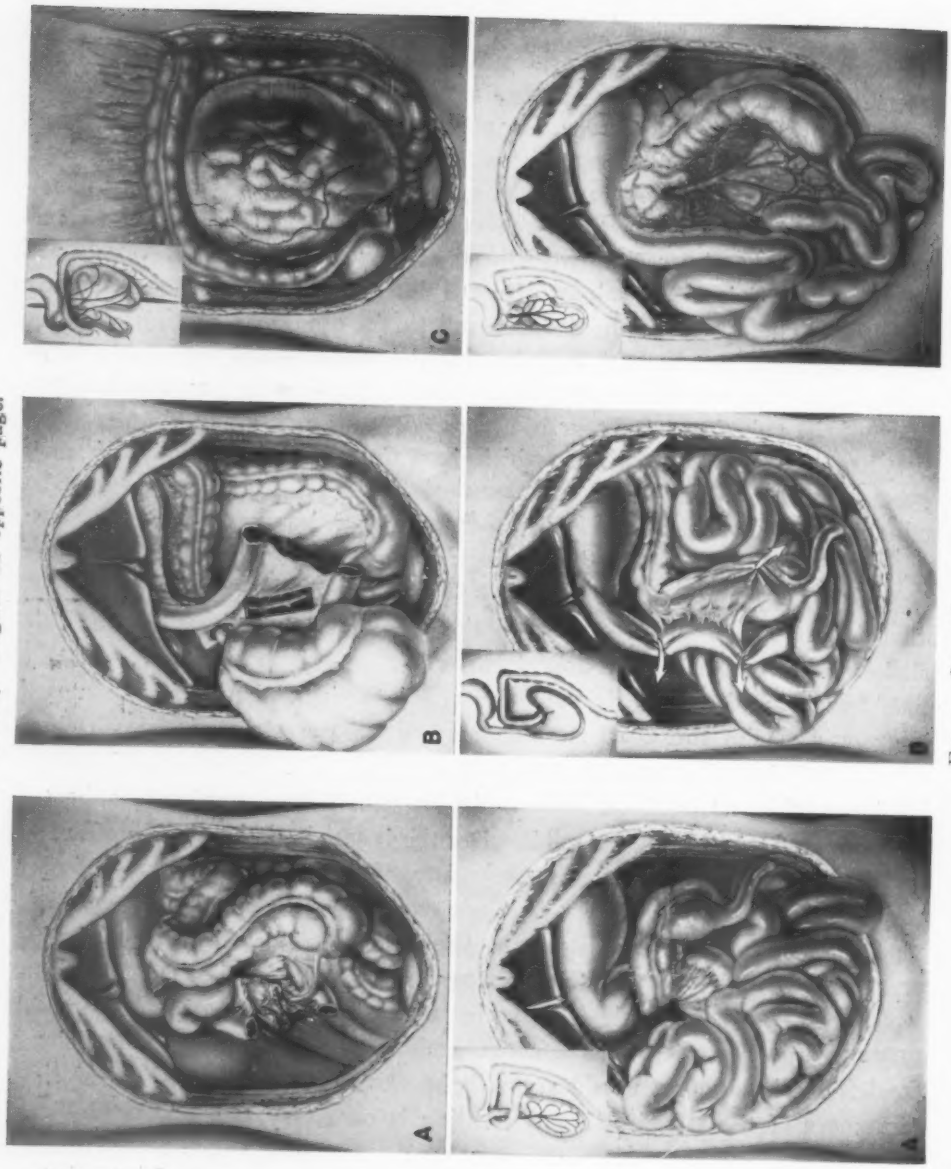

FIG. 4.—Legend on opposite page.

FIG. 5.—Legend on opposite page.

half of the abdomen (Fig. 5C). Following operation he took feedings without vomiting and was discharged, well, on the sixteenth postoperative day.

Case 3.—*Volvulus of Midgut.* B. T., No. C-47820, a 5-day-old Negro male, had a story of vomiting of yellowish material after each feeding since the second day of life. On examination he was dehydrated and weighed 2720 Gm. The outline of a distended stomach was visible in the epigastrium. Roentgen rays (Fig. 6B) showed the stomach full of air and practically no air in the small intestine. At operation exactly the same situation as in Case 1 was found. Following operation he did poorly, became jaundiced and failed to retain feedings well. The operative wound became infected. He died on the twentieth postoperative day.

Case 4.—*Volvulus of Midgut.* J. W., No. C-63611, was a 9-day-old white male. He had vomited since birth and at the age of 4 days had been operated upon elsewhere for pyloric stenosis, but none was found. He continued to vomit biliary material after operation and barium given by mouth 24 hours before we saw him was retained in the stomach and first part of the duodenum (Fig. 6C). His weight on admission was 2553 Gm. At operation a 360° clockwise twist of the midgut loop was found. After detorsion, entering and emerging loops of bowel were found adherent, and there were also adhesions crossing the duodenum to the kidney fossa. These were all freed and the bowel returned to the abdomen in a position of nonrotation. Following operation he did not vomit, but took feedings slowly. He gradually gained in weight and strength and was discharged in good condition on his twenty-seventh postoperative day.

Case 5.—*Volvulus of Midgut.* J. B., No. C-64536, a 6-week-old Negro male infant who had been in excellent health since birth suddenly began having abdominal pain 12 hours before admission. Six hours later he had a bowel movement consisting almost

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FIG. 4.—Abnormalities of the second stage of intestinal rotation.

(A) Nonrotation. The midgut loop has not rotated, the disposition of the viscera being essentially the same as at the end of the first stage of rotation (Fig. 1B) except that the bowel has returned to the abdominal cavity. The colon is entirely on the left and the small bowel on the right of the abdominal cavity.

(B) This shows reversed rotation. A 180° clockwise instead of a contraclockwise rotation during the second stage has occurred. The transverse colon thus comes to lie under the superior mesenteric artery while the duodenum passes in front of it. Subsequent fixation of the mesenteric root has trapped the colon under it.

(C) This shows internal hernia (Case 6). Most of the small intestine is enclosed in a peritoneal sac. The anomaly occurs during the second stage of rotation. When the midgut is reduced from the temporary umbilical herniation the bowel bulges into its own post arterial mesentery (insert Fig. 4C) instead of entering the free peritoneal cavity as it should (Fig. 3B). The wall of the sac is thus the mesentery of the terminal ileum and ascending colon.

FIG. 5.—Volvulus of the midgut (Case 2). Drawings made from sketches and photographs at the operation.

(A) Appearance on opening the abdomen of a 2-month-old infant with signs of chronic duodenal obstruction since birth. A 360° clockwise rotation of the midgut loop about the mesenteric root has occurred. The duodenum is obstructed at the volvulus and is dilated above it. Small bowel loops are not distended.

(B) Appearance on reduction of the volvulus. The mesentery and bowel of the entering loop of jejunum and emerging loop of colon are adherent. Sometimes adhesive bands also cross the duodenum in the region of the upper arrow.

(C) Appearance after separation of adherent bowel and mesentery of entering and emerging loops of the volvulus. Veins are distended and mesenteric lymph nodes enlarged by the chronic obstruction. The bowel is replaced in the abdomen in this position at the termination of the operation. The duodenum descends on the right of the vertebral column and all small bowel is on the right. The colon is entirely in the left side of the abdomen. This is the position of nonrotation.

FIG. 5.—Legend on opposite page.

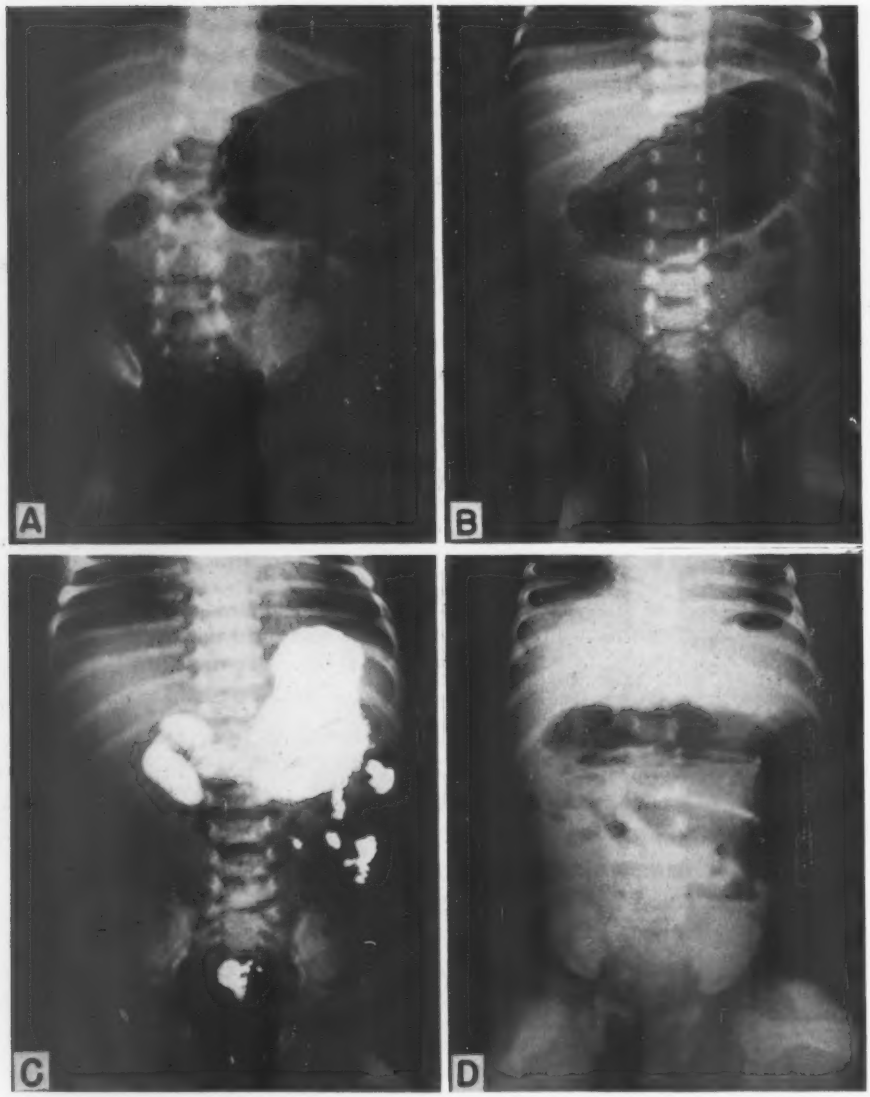

FIG. 6.—Roentgen rays of the abdomen in four infants with volvulus of the midgut is shown. (A)—Case 2. A 2-month-old infant with signs of chronic duodenal obstruction since birth. The stomach is distended with air and there is some gas in the bowel. (B)—Case 3. A 5-day-old infant with a story of biliary vomiting since birth. The stomach is distended with air. There is also a little gas in the colon, probably introduced by enema. (C)—Case 4. A 9-day-old infant with vomiting since birth who had been operated upon elsewhere on the fourth day for pyloric stenosis, but none was found. He was given barium by mouth 24 hours before being sent to us. This roentgen ray shows 24-hour retention of barium in stomach and duodenum with passage of a small amount of barium into the intestinal tract. (D)—Case 5. A 6-week-old infant with signs of acute intestinal obstruction of 12 hours duration. He had had three bloody bowel movements and was thought to have an intussusception before operation. The roentgen ray shows dilated loops of bowel full of gas in the left upper abdomen.

entirely of blood. Subsequently he had 2 more bloody bowel movements. He vomited several times. On admission 12 hours after onset of pain the child was lethargic and acutely ill. The abdomen was distended. No peristaltic sounds were heard. Roentgen rays showed dilated loops of bowel in the left upper abdomen (Fig. 6D). The stomach contained only a tiny gas shadow. A preoperative diagnosis of intussusception was made and immediate operation done. At operation volvulus of the entire midgut 540° in a clockwise direction was demonstrated. Bowel in the volvulus was of good color. There was a little chylous fluid in the peritoneal cavity. Lymphatics in the mesentery were dilated. Detorsion and division of adhesions extending from midtransverse colon across duodenum to right flank were divided and the bowel returned to the abdomen in the position of nonrotation. His postoperative course was satisfactory and he was discharged on the sixteenth postoperative day.

Malrotation. Innumerable irregular defects in intestinal rotation and fixation during the second stage are possible and are grouped together under the term malrotation. They are not difficult to understand, or to unravel at operation, if the normal stages of intestinal rotation are kept in mind.

If fixation of the small bowel mesenteric root over a wide base from Treitz fossa to the right iliac fossa fails to occur, volvulus of the entire mesentery is possible. Or the mesenteric root during the process of its fixation may trap a loop of bowel under it and give the appearance of a mesenteric defect. Other abnormalities of intestinal fixation may kink or compress the lumen of the intestinal tract at any level. These have been described most frequently in the duodenum. Ladd[14] has stressed the importance of inspecting the duodenum in all cases with abnormalities of rotation in order that anomalous bands may be relieved. In other cases the nonrotated cecum may be firmly fixed to, and may partially occlude, the duodenum or small intestine. Or the intraperitoneal bands and adhesions which occur regularly with anomalies of intestinal rotation may unite various combinations of intra-abdominal organs and be the cause of obstructive symptoms.

The inherent possibilities of irregularities of rotation and fixation are unlimited. Symptoms caused by them, if any, may be equally as varied but are usually those of partial or complete obstruction from volvulus or adhesive bands. Usually the true nature of the condition present is recognized only at operation. Waugh[22] emphasizes the "unusualness" of the symptoms, the "emptiness" of the right iliac fossa as a result of absence of the cecum from its normal position, and the roentgen findings after a barium meal as important findings which enable him to make the correct preoperative diagnosis in four of his five cases.

Internal Hernia. Complicated explanations of the origin of paraduodenal, retroperitoneal or internal herniations occur in the literature[18] which describe them as occurring in any one of as many as nine fossae about the duodenojejunal junction and four in the cecal area. Their explanation on the basis of an anomaly of intestinal rotation is much simpler and was first offered by Andrews[1] in 1923. He explained the origin of the paraduodenal hernia as imprisonment of small intestine under the mesentery of the right colon during the process of fixation of the midgut loop after its rotation.

Haymond and Dragstedt[11] in careful autopsy dissections of a case with large internal hernia previously observed at operation, showed that the abnormality was one of malrotation during the second stage of intestinal rotation. The essential feature of the abnormality was a rotation of the bowel into the mesentery of its postarterial segment (later to become the mesentery of the ascending colon (inset Fig. 4C) instead of into the free peritoneal cavity, during the phase of intestinal rotation when the midgut loop was reduced back into the peritoneal cavity from the umbilical orifice. In their case and in ours (Fig. 4C) almost the entire small intestine was in the sac. The amount of bowel involved may vary from a short loop to the entire small intestine.

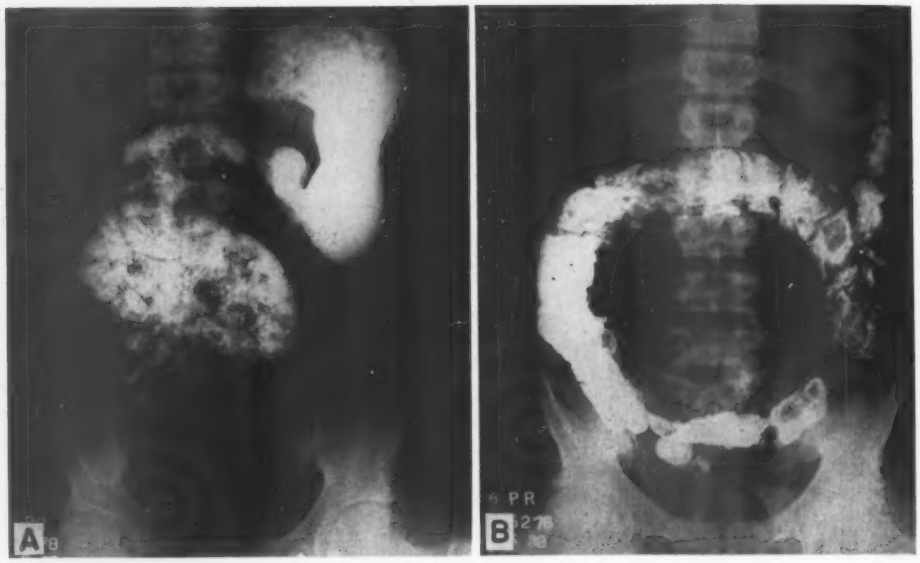

FIG. 7.—Roentgen rays of the barium filled stomach and intestinal tract in a patient (Case 6) with large internal hernia. (A) The small intestine appears to be enclosed in a circular pocket in the center of the abdomen. (B) This pocket is surrounded by colon of the midgut loop (right half of the colon). The splenic flexure and descending colon (hindgut) are normally placed.

Many of the patients with internal hernia have no symptoms and the condition is recognized only incidentally at operation, anatomic dissection or autopsy. If symptoms occur they are usually those of partial or intermittent intestinal obstruction.

In our case precise diagnosis of the condition present was made by our X-ray Department[19] who visualized the barium-filled small intestine enclosed in a circular pocket in the center of the abdomen (Fig. 7A). This pocket was surrounded by colon of the midgut loop (right half) (Fig. 7B). The splenic flexure and descending colon were normally placed.

At operation an adequate exposure, knowledge of embryonic origin, and delivery of as much bowel as is necessary will usually make the situation

clear. Since the wall of the sac is the mesentery of the postarterial segment of the midgut loop (terminal ileum and right colon) care must be taken not to damage its blood vessels. The recommended procedure is to withdraw the bowel from the internal hernial sac and to close its mouth with as many sutures as are necessary.[15]

Case 6.—*Internal Hernia.* J. M., No. A-5278, a 28-year-old Negro male, was admitted with a story of 5 attacks of cramping periumbilical pain during the preceding 2 months. Each attack was precipitated by large meals and was accompanied by vomiting of the food eaten at the preceding meal. Each attack subsided after 6 or 8 hours, during which he usually had to have a hypodermic injection. The physical examination showed nothing of significance. Roentgen rays (Fig. 7) showed the small intestine apparently to be enclosed in a round sac in the midabdomen, which was surrounded by the right half of the colon. At operation almost the entire small intestine was enclosed in a peritoneal sac (Fig. 4C) whose walls formed the mesentery of the right half of the colon. The small bowel could be withdrawn from the sac without difficulty, after which the opening of the sac was occluded by suture. Postoperative course was uneventful.

Reversed Rotation. In this condition a clockwise instead of a contraclockwise rotation of 180° occurs during the second stage. The transverse colon thus comes to lie under the superior mesenteric artery and the duodenum above it (Fig. 4B). If normal fixation of the root of the mesentery toward the right iliac fossa takes place with the bowel in this position the transverse colon becomes trapped in a tunnel beneath this acquired attachment. Fixation of the cecum and ascending colon is usually incomplete and torsion of the mobile right half of the colon with obstruction of the transverse colon at the site of the tunnel through the root of the mesentery may occur.

True reversed rotation is not common. McIntosh and Donovan[17] collected 16 cases and added one of their own. We have never had such a case. The illustration (Fig. 4B) is of Dott's patient, a 68-year-old man with symptoms of acute colonic obstruction of three days duration.

THIRD STAGE OF MIDGUT ROTATION

Embryology. This stage is characterized by descent of the cecum from its subhepatic position to the right lower quadrant and fixation of the mesentery of cecum and ascending colon in the right flank (Fig. 8B). Fixation of the descending colon and of the lower portion of the duodenum also occurs in this stage which is completed about the time of birth.

The important feature of this stage is the fixation of the ascending colon and cecum with its mesentery in the right iliac fossa and with it the fixation of the root of the small bowel mesentery on a wide base from left upper to right lower quadrants. The midgut loop originally dependent from a narrow pedicle at the origin of superior mesenteric artery now acquires a broad oblique attachment to the posterior abdominal wall. It is the absence of this attachment which predisposes to volvulus of the entire mesentery.

Anomalies of the third stage include subhepatic cecum, retrocecal appendix and mobile cecum.

Subhepatic Cecum. If the cecum fails to elongate and descend into the right iliac fossa it remains in a subhepatic position (Fig. 9A). If with the cecum in this position there is also failure of fixation of the small bowel mesentery on a broad attachment to the posterior abdominal wall, volvulus of all of the small intestine may occur. The only other significance of this anomaly is that appendicitis, if it occurs in such a position, may be unrecognized if the possibility of a high lying cecum is not kept in mind.

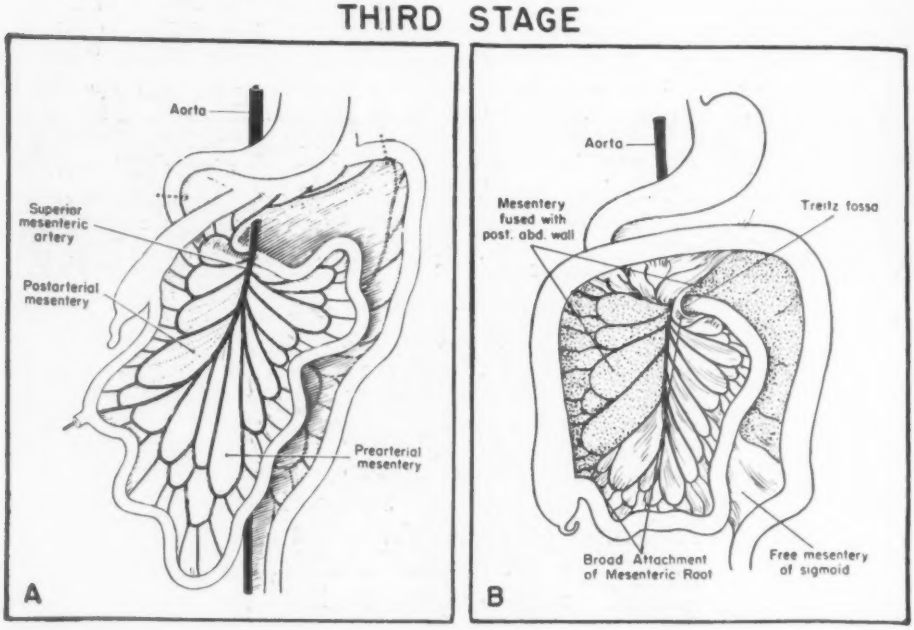

FIG. 8.—Third stage of intestinal rotation. This stage is characterized by descent of the cecum for its subhepatic position (A) to the right lower quadrant and fusion of its mesentery with the peritoneum of the right flank (B). The important feature is the fixation of the cecum and ascending colon in the right flank which gives a broad attachment of the root of the small bowel mesentery from Treitz fossa to right lower quadrant. The midgut loop originally dependent from a narrow pedicle at the origin of the superior mesenteric artery now acquires a broad oblique attachment to the posterior abdominal wall. It is the absence of this attachment which predisposes to volvulus of the entire mesentery. This stage is completed at birth.

Retrocecal Appendix. If the appendix is drawn under the cecum as the cecum descends into the right lower quadrant it may become fixed in a retrocecal position during the normal process of fixation of the cecum and ascending colon to the peritoneum of the right flank. This is a rather common anomaly and is well recognized by surgeons, although they usually fail to consider it an anomaly of rotation.

Mobile Cecum. Failure of fusion of the cecum and ascending colon and its mesentery to the peritoneum of the right iliac fossa in a normal manner allows

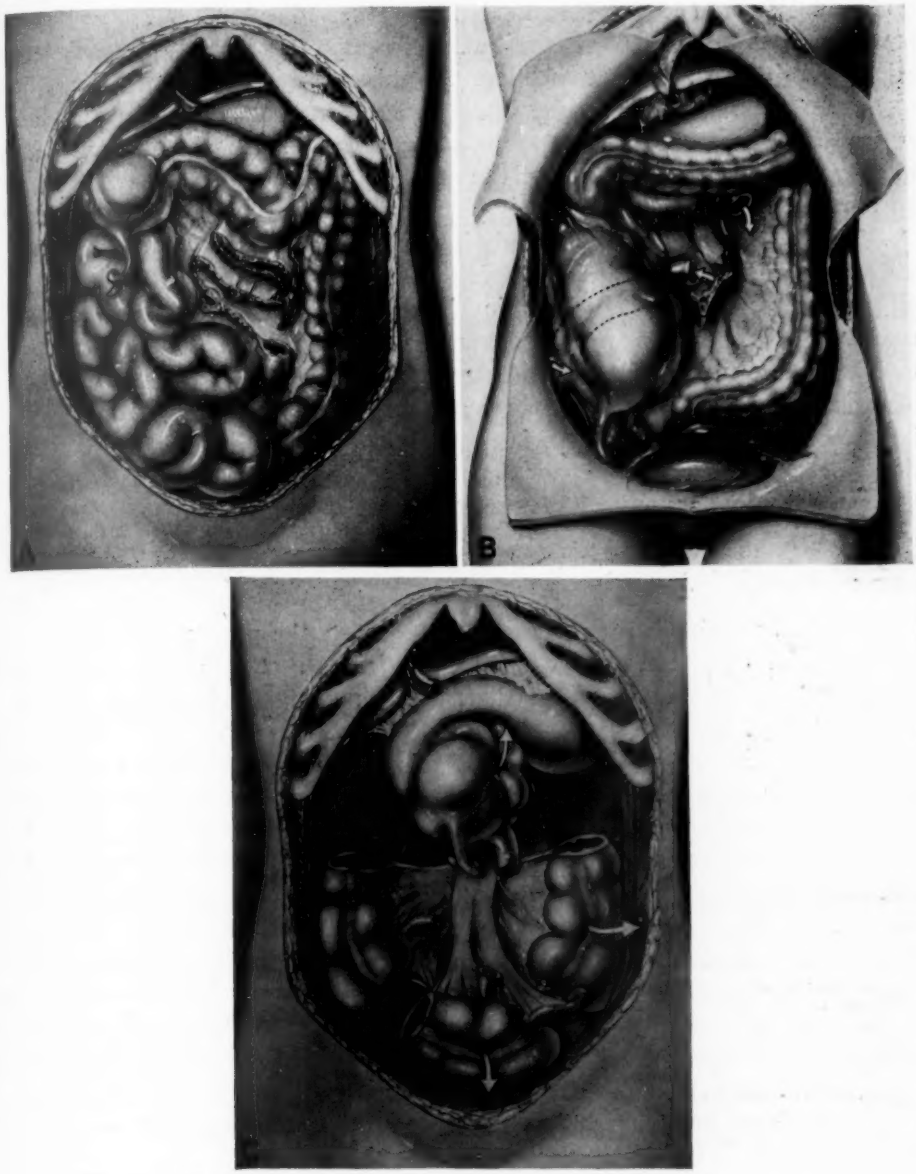

FIG. 9.—Abnormalities of the third stage of intestinal rotation.

(A) Subhepatic cecum. The cecum is arrested in the subhepatic position as at the end of the second stage of rotation. Since fixation of cecum and ascending colon in the right flank has not occurred, the mesentery of the small intestine fails to attain a broad fixation to the posterior abdominal wall from left upper to right lower quadrants and remains suspended from a narrow pedicle at the origin of the superior mesenteric artery. This may predispose to volvulus of the entire small intestine.

(B) Volvulus of the cecum. The cecum and ascending colon are mobile and have twisted 180° in a clockwise direction about the long axis of the ascending colon. The cecum is distended. Small bowel also usually becomes distended. Barium introduced by rectum would show a block at the hepatic flexure.

(C) Mobile cecum. Four hypothetical positions which a mobile ileocecal segment may occupy within the abdomen are shown. The mobile cecum may predispose to volvulus of the cecum. Its most dangerous consequence may be from the anomalous position of the appendix.

undue mobility of the cecum and ascending colon (Fig. 9C). This was formerly in itself thought to be the cause of symptoms, and numerous operations were devised for its correction. At present a mobile cecum is thought to be of significance only because it may be the seat of volvulus of the cecum or because it may allow the appendix to occupy a position almost any place in the abdomen so that appendicitis in an anomalously located appendix may go unrecognized.

Volvulus of the cecum occurs only in a mobile cecum. The twist is usually a longitudinal one about the long axis of the ascending colon[4, 23] (Fig. 9B), although there may also be a rotation in the oblique axis so that the cecum comes to lie in the epigastrium or left upper quadrant. The signs and symptoms are those of acute intestinal obstruction, often with enormous dilatation of the cecum. Roentgenograms show evidence of dilated small bowel plus a large cecal gas shadow. Barium introduced by enema will show an obstruction in the region of the hepatic flexure. At operation detorsion is done. Cecopexy or cecostomy may be necessary. If the bowel is gangrenous it must be resected.

SUMMARY

Intestinal rotation is the process which converts the position of the intestinal tract of the early embryo from a straight structure suspended on a common dorsal mesentery in the sagittal plane to the position seen in the normal child at birth.

A precise knowledge of the stages of this process of rotation and fixation and of the abnormalities which may occur in each is essential to the surgeon lest he be confused when confronted by one of the anomalies.

Each of the more common anomalies which occur in the three stages of rotation is described. They include omphalocele, nonrotation, volvulus of the midgut or of the entire small intestine, various types of malrotation, internal hernia, reversed rotation, subhepatic cecum, mobile cecum, volvulus of the cecum and the retrocecal appendix.

Some of the anomalies may exist without symptom and be detected only incidentally on roentgen ray examination, at operation, or when a diseased organ, particularly the appendix, is found in an anomalous position. If symptoms occur from the abnormality of rotation and fixation the usual ones are those of partial or complete intestinal obstruction.

Volvulus of the entire midgut or of the entire small intestine is the most common cause of intestinal obstruction secondary to anomalies of intestinal rotation. Symptoms depend upon the tightness of the twist and may be those of duodenal obstruction or of acute intestinal obstruction with or without signs of mesenteric vascular occlusion. The usual symptoms are partial or complete duodenal obstruction dating from birth. At operation evisceration, detorsion of the volvulus and release of adhesions uniting entering and emerging loops of bowel and of adhesive bands which may run across the duodenum is necessary. If these adhesions are not divided recurrence of the

volvulus may occur. The bowel is then returned to the abdomen in a position of nonrotation with the small intestine in the right abdomen and the colon on the left.

Omphalocele, amniotic hernia or exomphalos is the only anomaly in the first stage of rotation. The delicate umbilical cord structure covering such a hernia may rupture within the first few hours of life. To prevent evisceration it is necessary to close the defect as soon as possible after delivery.

Malrotation is the term used to designate the innumerable possible irregularities in rotation and fixation which may occur during the second stage. They include a wide variety of anomalous kinks, adhesive bands, and irregular fixations which are not difficult to unravel if the normal process of rotation is understood.

Paraduodenal, retroperitoneal or internal hernia is best explained as an irregularity of rotation wherein the midgut loop rotates into its own mesentery instead of into the free peritoneal cavity during the second stage. Since the wall of the sac is the mesentery of the ascending colon and terminal ileum its vessels must not be disturbed at operation.

Irregularities of descent and fixation of the cecum and ascending colon in the third stage are the most common anomalies of intestinal rotation. They include the subhepatic cecum, mobile cecum, and the retrocecal appendix. The most important feature of these abnormalities is that the appendix may be in an anomalous position.

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DISCUSSION—DR. ROBERT L. RHODES, Augusta, Ga.: I did not get the name of the author referred to by the previous speaker, but I rise simply to call attention to two things. One is the monumental contribution to this subject made by one of our own members and a former President. The opening comment of Doctor James E. Thompson in his Presidential Address "Surgery and Embryology" was very similar to the closing comment of our speaker. That is, if one notices and is familiar with these malformations, he does not become lost when he encounters them in the course of an operation.

This was a most excellent presentation, backed up with all types of roentgen rays which, of course, Doctor Thompson did not have the benefit of in 1919. I may say also that I presented a paper on this subject before the first Sectional Meeting of the American College of Surgeons in Birmingham in 1922, under the title of "Arrested Development of the Colon," the purpose being to emphasize what may happen, so that one will not be at a loss when faced with these conditions.

DR. J. W. DUCKETT, Dallas, Texas: There is probably no field of surgery in which the dictum of Halsted, of careful, gentle handling of tissues and complete hemostasis, is more important than in pediatric surgery; of course it is important in all surgery, but here it is essential to achieve such successful results as Doctor Grove and Doctor Gardner have reported in their series of cases. At the Dallas Children's Center we have had some experience with this type of lesion; actually, 15 cases to date since the Center was opened five years ago have grouped themselves into three series of five each. There were five cases of atresia of the duodenum, all of which were low, fortunately, and easily diagnosed early, and all were treated with duodenojejunostomy, as Doctor Grove has mentioned. However, the five atresias of the jejunum and ileum were all treated unsuccessfully, with death, as has been the case in the great majority of such lesions. Our five extrinsic type of obstruction have also been relatively simple, and all have survived.

I call this to your attention because of the fact that it has been universally found that atresias of the ileum and jejunum respond to surgery poorly. This is due to the fact that the atresias are usually multiple and extensive, and the distal segment of the bowel is often poorly developed; it may be hypoplastic or, if not, it is at least very small, collapsed and thin-walled, so that entero-anastomosis is extremely difficult. Even if it can be done technically, with an anastomosis resulting, function is often poor, as Doctor Gardner said.

Last year at Los Angeles I was impressed at finding that Doctor Norton Nichols at the Children's Memorial Hospital had had five successful cases in a row, of ileal and jejunal atresias, in which a very ingenious procedure was carried out. Disregarding Ladd's dictum of objection to enterostomy in a child of this age, but modifying it very ingeniously and adequately, Nichols has a series that promises to offer a new field for successful treatment of these cases. His procedure was, after exploration and finding the anomaly, to draw out both ends of the bowel through two stab wounds, on the left side as a rule, the proximal end being opened about 12 hours after operation and the distal end being opened at the time and a small tube sewed into the distal end. After the proximal end was opened a small cup made of cellophane or rubber dam was attached to the child's side by adhesive tape, and with careful nursing care and observation all the secretions from the proximal end were periodically collected and were immediately injected into the distal end through the tube that was sewed in. This served a double purpose; not only preserving fluids and electrolytes, the loss of which is serious in these small children, but also bringing about gradual dilatation of the distal segment, so that when the child had recovered satisfactorily, was beginning to gain weight and was being fed normally, a second operation was done about ten days after the first, at which time it was quite simple to produce a satisfactory functional enteroenterostomy. The results have been excellent so far in Doctor Nichols' series of unreported cases.

Dr. Deryl Hart, Durham, N. C.: First, I wish to confirm Doctor Grove's observation that at times these patients with duodenal atresia will gain weight and progress satisfactorily with a gastro-enterostomy, performed when the more desirable duodenojejunostomy is not possible. My first experience with this was in 1927, when I was a surgical resident. In a case of atresia of the duodenum a gastro-enterostomy was performed using the finest silk available. Only with the greatest difficulty could we obtain a suture line that would not leak at the site of each suture in the very small, thin-walled jejunum. The patient developed as satisfactorily as a child usually does following operation for congenital pyloric stenosis.

The second thing I wish to speak of are anomalies of intestinal rotation. Even though we have had a considerable number of these cases, and our resident staff members are familiar with the condition, on a number of occasions I have been called to the operating room to help the resident when he has been in trouble. At times he has realized the condition but was not sure as to the best way to handle it, but at other times he had not suspected the anomaly of rotation. There can be many variations of this condition. I think the most baffling one to me, to give an illustration, was a patient who had a non-rotation, then had a volvulus of the entire mid-gut, then had fixation of the cecum and ascending colon in its normal position. The volvulus must have occurred in embryonic life, since the cecum became fixed in its normal location after the volvulus had occurred. One of the best ways that I have found to make a diagnosis is to palpate the region of the duodenal-jejunal junction at the root of the mesentery and see if a tight band can be felt. This can be done without eviscerating the patient, but after the diagnosis is made evisceration is necessary before the volvulum can be untwisted. In the case mentioned above we could not eviscerate the patient because the cecum was fixed. The first step had to be mobilization of the cecum and ascending colon, so as to restore it to the condition of hanging on a pedicle of the superior mesenteric vessels. The mid-gut segment was then eviscerated and untwisted. It was then found that the intestine was in a position of non-rotation, and it was returned to the abdominal cavity in this position. The patient made a satisfactory convalescence.

Dr. J. M. T. Finney, Jr., Baltimore: I think we are particularly fortunate this morning to hear these two most interesting papers, and I certainly want to compliment Doctor Gardner on one of the most clearcut, beautiful demonstrations of both embryonic and anatomic development I have heard. I do want to call attention to one other application

of his demonstration of embryonic development in the normal rotation of the intestinal tract. That is one with which many of us have to deal nowadays, and which I am afraid is all too poorly understood by most surgeons—the question of the first, second and third portion of the duodenum. We very glibly refer to the retroperitoneal portion of the duodenum. Actually, there is no retroperitoneal portion, if one carries along with Doctor Gardner's demonstration of anatomic and embryonic development. It is rather a partial to complete fusion of two layers of the peritoneum as the gut rotates which makes it so easy, if one understands this, to mobilize the duodenum when one wants to get at the posterior portion of the head of the pancreas, or when one wants to do many of the operative procedures in the upper abdomen which are connected with a good many different types of operation nowadays. I have so often seen and heard surgeons have difficulty with the understanding of this question of mobilization of the duodenum that a demonstration such as Doctor Gardner has given this morning should be, I think, given widespread publicity in teaching medical students, because that is one thing that is, I believe, poorly understood.

Dr. Champ Lyons, New Orleans: Both these papers have so excited my admiration that I should like to express my appreciation to Doctor Grove and Doctor Gardner. The method of handling ileal atresia has been a matter of great interest to many surgeons. There is no question that a meticulous end-to-end anastomosis or entero-enterostomy, such as practiced by Doctor Grove, is infinitely preferable to the double-barrelled ileostomy. We have operated, however, upon two patients with gangrenous proximal portions of dilated bowel, by the technic suggested by Ladd and Gross of double-barrelled ileostomy closed immediately at the time of passage of the first succus entericus. This method has proved successful in both these instances and I believe it does have a place in cases with gangrenous perforation with peritonitis. Furthermore, Dr. Ralph Platou has urged us to abandon the use of barium by mouth in these children with intestinal obstruction, feeling that the regurgitation aspiration of barium contributes significantly to an increased incidence of pneumonitis. We have used lipiodol and found it satisfactory, and I should like to ask Doctor Grove what his feeling is about the preference of the use of lipiodol or barium in diagnosis of some of these more obscure lesions associated with intestinal obstruction in infants.

Dr. Lon Grove, Atlanta, Ga. (closing): I wish to thank Doctor Duckett and Doctor Lyons for their discussions. In answer to Doctor Lyons, we have not used lipiodol. We have used a very thin solution of barium when the gas pattern suggested duodenal atresia and the diagnosis could not be made without it. It should be used with great caution and, as stated in the paper, it should never be used when the air pattern suggests lower atresia and until we are certain that a tracheo-esophageal fistula is not present.

I again wish to emphasize the absolute necessity of close co-operation between the surgeon, the pediatrician and the roentgenologist.

Dr. Clarence E. Gardner, Jr., Durham, N. C. (closing): I wish to thank Doctor Rhodes, Doctor Hart and Doctor Finney for their very kind and cordial discussion. I appreciate Doctor Rhodes advising us of Doctor Thompson's contribution. I am sure we are all familiar with the work Doctor Rhodes has done in this field, as well as with atresias of the rectum. The whole problem is well known to surgeons of his generation but, as Doctor Hart stated, the younger men are not familiar with anomalies of intestinal rotation and are frequently confused, and that was the reason we were anxious to record again these findings which, of course, are not new.

I appreciate Doctor Finney's comments very much and I would like to elaborate further and say that knowledge of the normal process of rotation is not only utilized when the duodenum must be mobilized but also whenever the colon is mobilized for resection.

NON-MALIGNANT DUODENO-COLIC FISTULA*

REPORT OF TWO CASES

SIR HENEAGE OGILVIE, K.B.E., M.CH., F.R.C.S.

DUODENO-COLIC FISTULA is a lesion of great rarity. Most of the cases recorded have been due to the extension of a carcinoma of the transverse colon, but five cases of benign duodeno-colic fistula have been recorded since 1885. The cause is reported to have been duodenal ulcer in three cases, typhoid ulceration in one and ulcerative colitis in one.

Rees[5] in 1933 reported a fistula between the transverse colon and the duodenum just below the opening of the common bile duct, which had apparently arisen by the perforation of a typhoid ulcer in an attack 28 years before. The patient was a woman of 62.

Ormandy and Bargen[4] in 1939 reported that at autopsy on a patient dying of ulcerative colitis, multiple fistulas were found between the colon and the stomach and duodenum.

McPeak[3] in 1940 reported two cases. The first was in a priest, age 58, who had complained of looseness of the bowels and loss of weight for three years. The fistula connected the transverse colon just beyond the hepatic flexure with the duodenum at the junction of its second and third parts. At operation an active ulcer was found at the site of the opening in the duodenum. McPeak's second case was in a man of 46 years who, after 15 years of indigestion, had had an acute attack of pain thought to be due to perforation a year before, followed by diarrhea. At operation a communication was found in the same site as in the first case. McPeak refers to Rees' case, and to a doubtful one reported by Blondeau, Derrieu, and Miramond de la Roquette[1] in 1935 that was probably malignant.

McClinton[2] in 1944 added a fifth case, in a man, age 44, who had suffered for many years from duodenal ulcer. Two years previously he had had an acute attack of pain suggesting perforation, and after this suffered from constant diarrhea. At operation a hole the size of a thumb was found between the proximal end of the transverse colon and the duodenum two and a half inches beyond the pylorus.

CASE REPORTS

The two cases which I report were entirely different in their etiology and in the site of the fistula from those previously recorded.

Case 1.—In 1946 I was asked to see a lady of 52 years who had suffered from intermittent attacks of diarrhea since she was 18, and was known to have had a duodeno-colic fistula for at least 20 years, although the attacks were separated by intervals of normal health. She readily concurred with the advice given to her by her physician that the only danger she ran was that she might fall into the hands of a surgeon.

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949.

Six months before I saw her the diarrhea had become continuous and progressively worse. She lost weight rapidly, and when I saw her was almost a skeleton, weighing about 70 pounds, although she was a tall woman. She lost four more pounds in the next three days and I was forced to operate upon her as an emergency.

In view of the rapid change in the clinical picture, I expected that the fistula, whatever its original nature, had undergone a malignant change. However, after mobilizing the right colon, I found a short track joining the lowest part of the third portion of the duodenum to the back of the ascending colon a handsbreadth below the hepatic flexure. I divided the track and closed each end with a double purse string suture. Recovery was uneventful.

A collection of calcareous lumps round the track showed that the cause of the fistula was a caseating tuberculous gland that had ulcerated simultaneously into the two viscera. Age, constipation, and loss of weight had caused the right colon to sink down and drag on the fistula, so that latterly the duodenum had been pulled into a funnel, shooting all its contents into the colon.

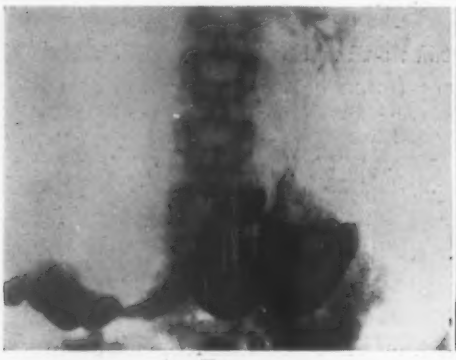

FIG. 1

FIG. 2

FIG. 1.—(Case 1.) Barium meal. Barium can be seen entering the ascending colon from the lowest part of the duodenum, which has been pulled out of line by the drag of the fistulous track.

FIG. 2.—(Case 1.) Barium meal, half an hour later. The whole of the first and second parts of the duodenum, and the ascending colon and hepatic flexure are now filled.

Case 2.—In June, 1949, a naval commander, age 54, was referred to me as suffering a gastrocolic fistula. He had been perfectly well till March 7, 1949, when he had an attack of diarrhea and vomiting. The diarrhea cleared up after ten days but recurred three weeks later. From that date the attacks of diarrhea became more frequent and he lost weight and strength. Since he had seen much service in the Far East with the navy, some form of dysentery was suspected, and a barium enema was ordered. The discovery of a communication between the ascending colon and (apparently) the pyloric end of the stomach surprised the radiologist as much as his medical attendant.

At operation, which was even easier than in the first case since the patient was not so ill, exactly the same state of affairs was disclosed. There was a fistula between the lowest part of the third portion of the duodenum and the back of the ascending colon three inches below the hepatic flexure, and the cause was a caseating gland. Closure was easy and recovery rapid. The patient, knowing I was going to discuss his case in America, wrote to me just before I left to tell me that he had put on 28 pounds in weight since the operation, and that he could now consume double gins with enjoyment and full therapeutic effect.

My reason for reporting these two cases is that they are examples of a lesion that has not, as far as I know, been reported up to the present time in

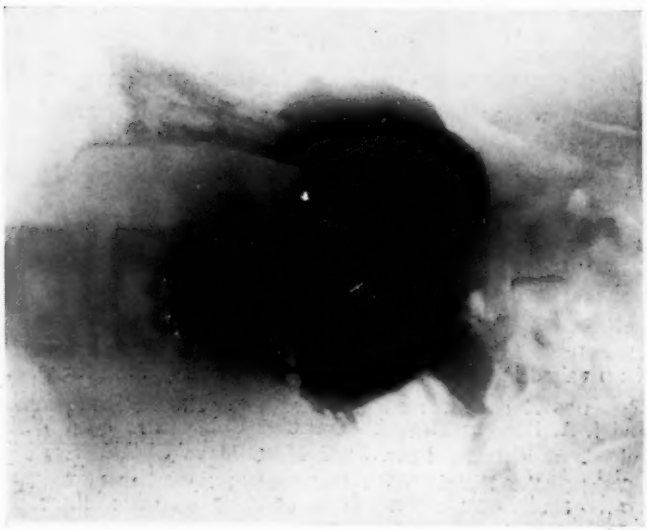

FIG. 4.—(Case 2.) Barium meal given two days later. Some barium has entered the ascending colon.

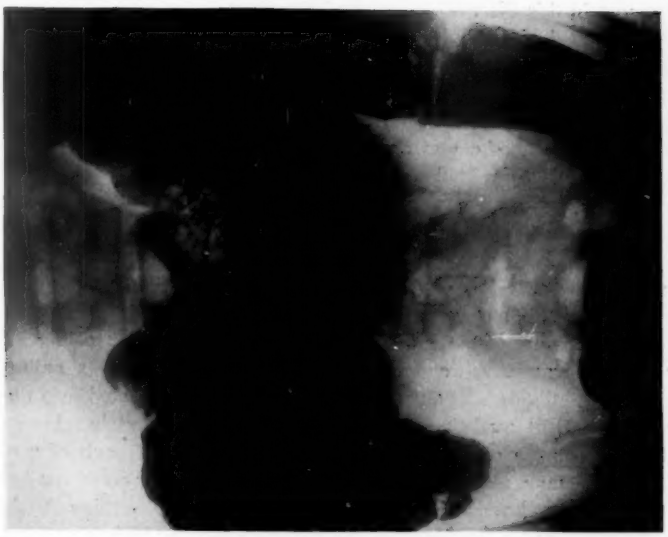

FIG. 3.—(Case 2.) Barium enema administered on the tentative diagnosis of dysentery. The barium has entered the duodenum, and a little has passed the pylorus into the stomach.

surgical literature. I have been told that the passage of a catheter in a patient with urinary retention due to an enlarged prostate gives a surgeon the biggest dividend in gratitude with the least outlay in time and skill. This is no longer true. The closure of a non-malignant duodeno-colic fistula beats it.

SUMMARY

Two cases of non-malignant duodeno-colic fistula are reported. In each case the communication was between the lowest part of the third portion of the duodenum and the back of the ascending colon, and appeared to have originated in a caseating tuberculous gland.

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DISCUSSION—DR. FRED KROCK, Ft. Smith, Ark.: After hearing Doctor Ogilvie's paper on the rarity of duodeno-colic fistulas, I think that I may be pardoned for including an additional case report in this discussion.

Two months previously I saw with one of my surgical colleagues, Doctor J. D. Olson, a 68-year-old white female who gave a history of gastro-intestinal complaints dating back to the age of 19 years. At that time she had noted indigestion, cramping, diarrhea and gas. Two years later nausea and vomiting were added to the discomfort which occurred periodically. At the age of 45 she was confined to bed for one month with an attack of bloody diarrhea. After this episode she noted that the vomitus had a very foul taste and that it had a fecal odor. During the succeeding years she had limited her diet very sharply in order to reduce the incidence of the attacks, and at times she noted undigested food in her stools.

Physical examination revealed only emaciation and dehydration. Roentgen ray examination demonstrated a fistulous connection between the descending portion of the duodenum and the hepatic flexure of the colon. A barium enema could be readily manipulated so as to enter the duodenum. At operation (J.D.O.), the fistula was exposed at the indicated site (slide) and divided. The two openings were easily inverted into their respective lumens, and the patient made an uneventful convalescence and has become a most grateful individual. It was our opinion that the fistula arose as the result of a perforating diverticulum of the duodenum, although a perforating duodenal ulcer could not be ruled out.

SIR HENEAGE OGILVIE, London, England (closing): I have nothing further to add except to say that I am glad one further case has been reported, and I believe Champ Lyons has another that we shall get later.

Again, I appreciate very much the intimate association of this group, and one thing I have learned today is the definition of a country doctor. In my early days I did a certain amount of surgery in the country and I had a different definition of myself. I had an old farmer who paid me in pound notes; I asked him if he would not give me a check, and he said, "No, this suits my income tax; I shall put you down as a load of manure."

PHEOCHROMOCYTOMA: ITS DIAGNOSIS AND TREATMENT*

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(By Invitation)

AND

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FROM THE DEPARTMENTS OF INTERNAL MEDICINE AND SURGERY, THE

PHEOCHROMOCYTOMA, an epinephrine producing tumor, although rare, does occur with sufficient frequency (approximately 200 reported to date) to warrant a suspicion of its presence in a patient having any type of vasomotor attacks or nonthyroid hypermetabolism or both. The suspicion of such a tumor plus the help of a few pointed clinical studies will lead to the correct diagnosis, and a carefully executed surgical procedure aided by one of the new adrenolytic drugs (dibenamine-benzodioxan) will bring about a cure. The patient with pheochromocytoma may be entirely asymptomatic prior to a fatal attack but it is with the "spell-inciting" or hypermetabolism-producing tumors that this paper deals. Four such cases have been observed during the last three years. These four cases present all of the ramifications of the clinical entity of pheochromocytoma and all four patients were successfully operated on.

During the three year period in which these four cases were diagnosed clinically and the tumors successfully removed, we have encountered two postoperative fatalities due to unrecognized pheochromocytomas. One patient died following lumbar sympathectomy for peripheral vascular disease; a severe hypertensive reaction developed during the course of the operation and was followed by acute heart failure and death. The second patient had an incisional hernia repaired; a similar reaction and death occurred. During this same period of time we know of two additional similar deaths in one of our hospitals, one during a thoracic operation and the second at the conclusion of a gastrectomy. In all four cases a pheochromocytoma was found at necropsy and was unquestionably the cause of death.

Should a hypertensive reaction occur during the course of any operative procedure it should lead to the suspicion of a pheochromocytoma. Based on our experience it must be emphasized that this reaction constitutes an acute emergency not unlike cardiac arrest, and one should be prepared to meet it, just as we have prepared ourselves to manage cardiac arrest. One of the following courses must be instituted.

1. Immediately stop the operation and after the patient's recovery, submit him to further investigative study.

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949.

2. If the operation is intra-abdominal, immediately explore the adrenals and sympathetic chain areas, and if a tumor is found, steps should be taken to proceed with its removal.

3. Use an adrenolytic drug to control hypertension and continue with the initial operation. Postoperative investigation should then be carried out to confirm the presence of a pheochromocytoma. (The response of the blood pressure to the adrenolytic drug is additional confirmatory evidence.)

In the light of our present knowledge and with the use of the new adrenolytic drugs, it now seems that one is justified in proceeding with exploration to make certain whether or not a pheochromocytoma is present.

Case 1.*—A 23-year-old woman gave as her complaint recurring attacks, 5 to 6 times daily and lasting one to 15 minutes during the previous 3 years, of weakness, severe throbbing headache, dyspnea, palpitation, epigastric and substernal pain, nausea and vomiting, unsteadiness and blotchiness of the skin. The attacks usually came after eating and at times were relieved by vomiting or sneezing. Between the attacks she was perfectly well. During the previous two years thyroidectomy, appendectomy and cholecystectomy had been performed and teeth removed in the hope of stopping the attacks.

The pulse rate was 120 and blood pressure 102 mm. systolic and 94 mm. diastolic. Shortly after admission to the hospital she had one of her typical attacks during which the blood pressure was 180 mm. systolic and 120 mm. diastolic. This led to the suspicion of a pheochromocytoma.

The blood sugar during an attack rose from 100 to 166 mg. per 100 cc. An intravenous pyelogram showed a depression of the left kidney. The basal metabolic rate was +9 when the blood pressure was 112 systolic and 80 diastolic, and +36 during a typical attack with the blood pressure 200 systolic and 110 diastolic. Histamine and mecholyl diagnostic tests were carried out and found to incite a typical attack with a systolic pressure of 200 and diastolic pressure of 140.

Exploration of the right adrenal area gave negative results. A tumor, 8 cm. in diameter, was found in the left adrenal area and was removed. During its removal a severe hypertensive reaction with circulatory collapse occurred, from which the patient recovered. The tumor weighed 260 Gm.; the pathologic diagnosis was pheochromocytoma.

The patient remained well for one year and then a swelling developed in the right side of the neck. Biopsy revealed this to be a metastatic pheochromocytoma. The blood pressure had remained normal and the mecholyl test was negative. Radical neck dissection was carried out, followed by roentgen ray treatment.

It is now three and a half years since the pheochromocytoma was removed and one and a half years since radical resection of the neck was performed. Her physician reports that the patient's health has failed gradually; she has severe neck pain which requires narcotics for relief, indicating advancing metastatic disease. She has had no further hypertensive attacks.

Case 2.†—A 24-year-old woman gave as her chief complaint blindness, which began five months before admission to the clinic, during the fourth month of pregnancy. The blood pressure was found to be elevated when the eye symptoms first began. During the sixth month of gestation convulsions developed for which she was hospitalized. She remained semicomatose for three weeks, then labor was induced and a dead fetus delivered. Recovery was slow; the blood pressure remained at a high level and there was no improvement in vision. Following discharge from the hospital she failed to regain her strength. She perspired at times and had frequent episodes of flushing and forceful pounding of the heart.

* Previously reported by Guarneri and Evans.

† Previously reported by Bartels and Kingsley.

On examination, the blood pressure was 180 systolic and 136 diastolic with a pulse rate of 110. A firm mass was found in the left side of the pelvis. The retinal vessels were markedly constricted and scattered areas of old exudate and pigmentary degeneration were seen.

Because the intravenous pyelogram disclosed slight delay in the emptying time of the right kidney, retrograde pyelography was undertaken. The blood pressure before induction of anesthesia with intravenous sodium pentothal was 120 systolic and 80 diastolic. Simultaneously with the injection of dye into the right ureter the blood pressure rose to 190 systolic and 150 diastolic, followed by profound circulatory collapse (Fig. 5). Recovery gradually took place during the next five hours.

Following this procedure, the blood pressure was found to fluctuate widely. It was not unusual to obtain a reading of 120 systolic and 80 diastolic and then within a minute or two the patient flushed and perspired and the blood pressure climbed to a level of 200 systolic and 150 diastolic. Because of the extreme variability of the blood pressure and inability to obtain basal readings of sufficient duration to permit the performance of specific diagnostic tests, it was decided to carry out the necessary tests under avertin anesthesia. The histamine test was negative but the mecholyl and etamon tests were positive.

Since the diagnosis of pheochromocytoma seemed certain, it was decided to explore the lower part of the abdomen to determine the nature of the pelvic tumor and then palpate the adrenal areas. The left ovary was found to be involved in a cystic mass, and this was removed. Then palpation revealed a tumor mass in the region of the left adrenal gland. This tumor was successfully removed by retracting the stomach, colon and pancreas. During the operation the blood pressure remained normal. The pathologic diagnosis was pheochromocytoma.

On the second day after operation the patient became confused, lethargic and word aphasic. This cleared in 10 days. A lumbar puncture disclosed clear yellowish fluid under increased pressure; the total protein was 55 mg. per 100 cc.

On the fourteenth postoperative day the histamine and mecholyl tests were negative.

Six months after operation the patient was in excellent health except for visual impairment and slight residual word aphasia. The blood pressure was 124 mm. systolic and 80 mm. diastolic. She has had no further attacks.

Case 3.*—A 30-year-old married woman complained of attacks of substernal distress and palpitation for 10 years, profuse perspiration and lifetime intolerance to heat. Her illness actually began at the age of 9 years when, because of excessive perspiration and 30 pound weight loss, she was suspected of having hyperthyroidism. She was given roentgen ray treatment to the thyroid gland. Because of sugar in the urine, insulin was given for a time. At the age of 20 she became pregnant and because of hypertension (200) and albuminuria, a therapeutic abortion was done. The blood pressure fell to normal. Following the pregnancy she began to have attacks of substernal pain with radiation into the arms and neck, lasting from several minutes to an hour. These attacks were associated with fear of impending death, profuse perspiration, marked palpitation, dyspnea and oftentimes a severe occipital pounding. The attacks were followed by prostration which lasted for hours.

When the patient was 22 years of age, because the basal metabolic rate was found to be +60, a subtotal thyroidectomy was performed. This did not relieve the symptoms, which were diagnosed as angina. The blood pressure was never taken during an attack.

At the age of 30, because of continued attacks, intolerance to heat, excessive perspiration and an elevated basal metabolic rate, +30 and +41, the patient was referred to the Lahey Clinic.

The blood pressure varied between 180 and 110 mm. systolic and 100 and 80 mm.

* Previously reported by Bartels and Arnold.

diastolic. No thyroid remnant was palpable. She perspired periodically, and with this there was a slight rise in the pulse and blood pressure. The urine did not reveal sugar but the glucose tolerance test indicated diabetes. A roentgenogram of the chest showed arteriosclerotic changes in the arch of the aorta. An intravenous pyelogram demonstrated calcification about the upper pole of the right kidney.

Histamine, given intravenously, produced some increase in the systolic and diastolic blood pressure but did not incite an attack. The epinephrine test produced a rise in both the systolic and the diastolic pressure and symptoms similar in every respect to those of the mild attacks. The mecholyl test produced a rise in blood pressure to 190 mm. systolic and 110 mm. diastolic but did not produce an attack.

When the tests were carried out under intravenous pentothal, the histamine and mecholyl tests gave negative results. Under avertin anesthesia, intravenous etamon produced a fall in the blood pressure but there was no overshoot. All efforts to provoke an attack were unsuccessful.

The basal metabolic rates were +60, +58, +62 and when taken under pentothal were +53 and +60.

Since the available evidence, in spite of negative provocative tests, favored a pheochromocytoma, exploration was deemed advisable. The left adrenal area was first explored and found to be normal. Exploration of the right side revealed a large tumor arising from the right adrenal gland.

During the operation, palpation of the tumor produced a rise in the blood pressure to 180 systolic and 126 diastolic with a rise in the pulse of 170. Dibenamine (60 mg.) was given at this time; the blood pressure returned to 120 systolic and 90 diastolic but the pulse remained 140. The tumor was removed. It weighed 75 Gm. and microscopically was found to be a pheochromocytoma.

After operation all of the patient's complaints were relieved and have not returned (10 months) and for the first time in her life she has experienced chilliness. The blood pressure and basal metabolic rate were normal, as were the glucose tolerance test, histamine, mecholyl and cold pressor tests.

Case 4.—A 20-year-old girl was well until 8 months before being seen, when morning occipital headaches began. One month later she noted continuous excessive sweating and palpitation at night. In the seventh month of her illness the blood pressure level was found to be very high, and the basal metabolic rate was elevated (+37 and +40). Because of the suspicion of thyroid disease she was referred to the Lahey Clinic for study.

Physical examination revealed the skin to be warm and moist. The pulse rate was 120 and the blood pressure 200 systolic and 130 diastolic. The thyroid was not palpable.

The glucose tolerance curve was typically diabetic. An intravenous pyelogram showed very slight downward displacement of the left kidney. The dibenamine test was positive, the blood pressure falling to a normal level (130 systolic and 90 diastolic) during the intravenous administration of 182 mg. of dibenamine. The basal metabolic rate was +42.

The hypermetabolism without hyperthyroidism, the diabetic glucose tolerance test and the positive dibenamine test seemed to confirm the diagnosis of a pheochromocytoma and the slight downward displacement of the left kidney placed the tumor on the left side.

Exploration of the left adrenal area revealed the tumor, but before it was removed the right adrenal area was explored by opening the peritoneal cavity, and found to be normal. The tumor, a cystic mass, was then removed. It measured 6 cm. in diameter. On microscopic study it was found to be a paraganglioma. During the course of the operation dibenamine was given intravenously, which maintained the blood pressure at a subhypertensive level; a total of 175 mg. was given.

The patient's postoperative course was satisfactory. The blood pressure was normal and all symptoms were relieved. A follow-up examination 6 weeks after operation revealed a normal blood pressure, a normal glucose tolerance test and a basal metabolic rate of +10.

SYMPTOMS

The duration of symptoms in two cases was four to eight months, in one case three years and in one case 20 years. Attacks or "spells" with vasomotor manifestations constituted the outstanding symptoms (Fig. 1) in three of our four cases. Palpitation was noted in all four cases and sweating was periodic in two and constant in two patients. Two patients suffered from substernal pain with dyspnea during an attack which had the characteristics of angina pectoris. Occipital headache was noted by two patients. Blotchiness of the skin was observed by one patient during a typical attack. Blindness, developing during the early months of pregnancy, was the chief complaint of one patient. Three of the four patients, because of known hypermetabolism, were previously considered to have thyroid disease. Two of these patients (Cases 1 and 3) had been submitted to subtotal thyroidectomy without benefit, and the third patient was sent to the clinic for a thyroidectomy.

	CASES 1	2	3	4
A. CARDIOVASCULAR ATTACK	+	+	+	–
B. SWEATING		+	+	+
C. BLOOD PRESSURE				
1. FLUCTUANT	+	+	+	
2. SUSTAINED				+
D. ELEVATED B.M.R.	+		+	+
E. DIABETIC G.T. TEST	+(‡)		+	+
F. ABNORMAL I.V. PYELO.	+		+	+

(‡) ELEVATED BLOOD SUGAR DURING ATTACK

FIG. 1.—Essential symptoms and clinical findings in Cases 1 to 4.

CLINICAL FINDINGS

One patient (Case 4) had sustained hypertension, two patients (Cases 1 and 2) were observed during typical hypertensive attacks and one patient (Case 3) had mild fluctuant hypertension.

Three patients were studied from the standpoint of the basal metabolic rate. Case 1 (Fig. 2) had a normal metabolic rate when the blood pressure was normal but the rate was +35 when taken during an hypertensive attack. Case 3 had metabolic rates ranging around +60 during periods of near normal blood pressure. Even under pentothal anesthesia the rate remained elevated, +55. Case 4 had sustained hypertension, the basal metabolic rate ranging from +35 to +42. Case 3 illustrates that the basal metabolism can be elevated even during periods of normal blood pressure.

Intravenous pyelograms were sufficiently abnormal in three patients to indicate the probable site of the pheochromocytoma. In two cases slight downward displacement of the kidney gave the clue and in one patient there was calcification of the tumor.

Abnormal sugar metabolism was observed in three patients. Case 1 had a rise in the blood sugar of from 100 to 166 mg. during an hypertensive attack.

Cases 3 and 4 had suggestive diabetes glucose tolerance tests (Fig. 3). Glycosuria was not a feature of any of the cases.

SPECIFIC DIAGNOSTIC TESTS*

1. Histamine.[9] Case 1 developed a typical hypertensive attack following the intravenous administration of histamine (0.05 mg.). Case 2, because of the ease of attacks and our failure to obtain a near normal base line of blood pressure for study, was given histamine when under pentothal anesthesia (Fig. 5B). This test gave negative results. Case 3 also had a negative histamine response under pentothal anesthesia (Fig. 6B). When the test was carried out in the usual way there was a very slight blood pressure rise (Fig. 7A) but an attack was not incited and the systolic pressure rose only to 180 and the diastolic to 110. These experiences with histamine indicate that this drug is not a specific test for pheochromocytoma since an attack may not be incited by its use and also, pentothal anesthesia may nullify its effect. The explanation for this failure of histamine to cause a reaction while the patient is under pentothal is not apparent but it does suggest that histamine acts

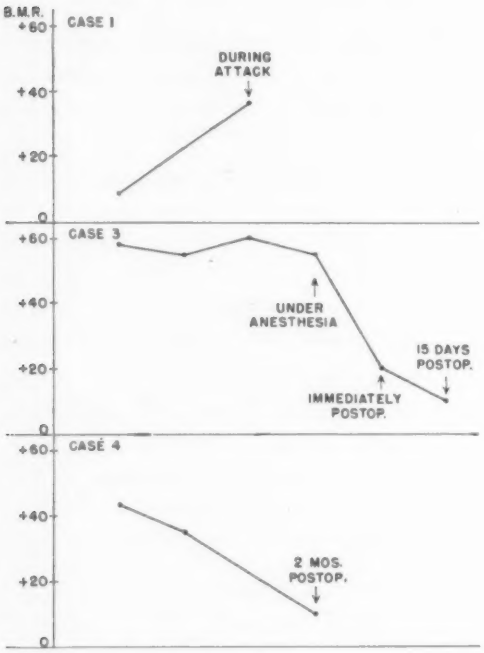


FIG. 2

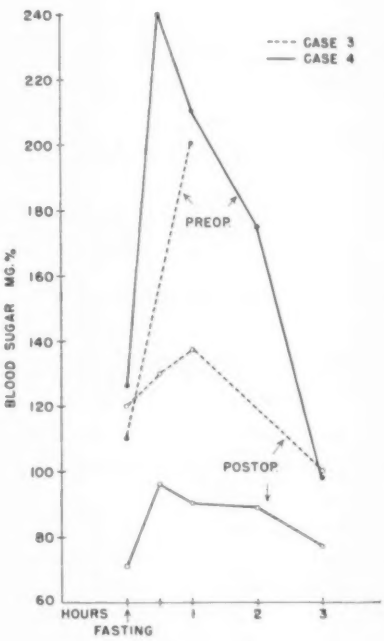


FIG. 3

FIG. 2.—Basal metabolic rates before and after removal of pheochromocytoma in Cases 1, 3 and 4.

FIG. 3.—Glucose tolerance tests before and after removal of pheochromocytoma in Cases 3 and 4.

* See Figure 4.

through the emotional center in stimulating the pheochromocytoma since histamine given intravenously does produce very unpleasant head symptoms.

2. Mecholyl.[7] In Case 1 a characteristic hypertensive attack followed the subcutaneous administration of 15 mg. of mecholyl. Case 2 was given mecholyl while under pentothal anesthesia (Fig. 5B), with a characteristic pheochromocytoma response (systolic pressure 210, diastolic 150, a diastolic hypertensive response). Case 3 (Fig. 7C) had some rise in the blood pressure which was very gradual but it did not greatly exceed the basal blood pressure and the patient did not experience symptoms such as she had during her attacks. When the test was done under pentothal anesthesia the test (Fig. 6B) was completely normal; a sufficient fall took place following the injection but no abnormal rise followed.

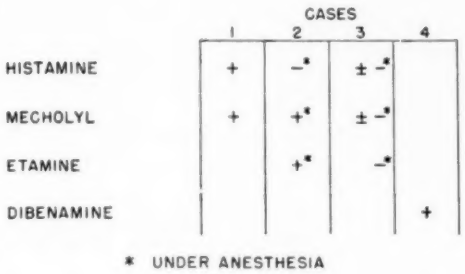


FIG. 4.—Specific diagnostic tests in Cases 1 to 4.

These experiences with the mecholyl test indicate that it is a diagnostic aid since in the first two cases it was positive. In the third case, however, it was negative, indicating that it is not a specific test for pheochromocytoma.

3. Etamon.[8] In Case 2, 3 cc. of etamon was given intravenously (Fig. 5C), under avertin anesthesia. It produced a severe, almost precipitous systolic and diastolic hypertensive response which, after the initial rise, became wavelike during the next 20 minutes. In Case 3, when the etamon was given while the patient was under avertin narcosis (Fig. 6C) the test was negative; a prompt fall occurred following the administration of etamon but no subsequent abnormal rise took place. As with histamine and mecholyl, etamon is not a specific incitatory hypertensive agent in the presence of a pheochromocytoma and a negative response does not rule out the presence of a tumor.

4. Dibenamine.[11] Because of sustained hypertension dibenamine was used as a diagnostic aid in Case 4. After the blood pressure (Fig. 8) had reached a base line of 190 systolic and 120 diastolic, a 0.6 per cent solution of dibenamine was begun intravenously. After 15 minutes the systolic pressure fell to 140, with a slower fall in the diastolic pressure, reaching 90 only after 30 minutes. The lowest blood pressure was reached at one hour, on termination of the test, when the systolic pressure was 120 and the diastolic 80. A total of 182 mg. of dibenamine was administered. This test was strong evidence for a pheochromocytoma as reported by Spear and Griswold.[11] Benzodioxan,[5] another adrenolytic agent, has been reported to be an equally informative agent but its use may cause an alarming hypertensive reaction in patients who do not have a pheochromocytoma.[4]

Since most of the previously mentioned studies are carried out on hypertensive patients in preparation for sympathectomy, we have been able to rule out the possible presence of pheochromocytoma in this type of patient. Smithwick has reported pheochromocytoma to be a finding in 0.5 per cent of his cases of hypertension in which sympathectomy is performed.

SURGICAL TECHNIC

Operation for pheochromocytoma is best carried out under general anesthesia utilizing ethylene-ether or cyclopropane-ether administered through an endotracheal tube. Spinal anesthesia should be avoided because of its direct

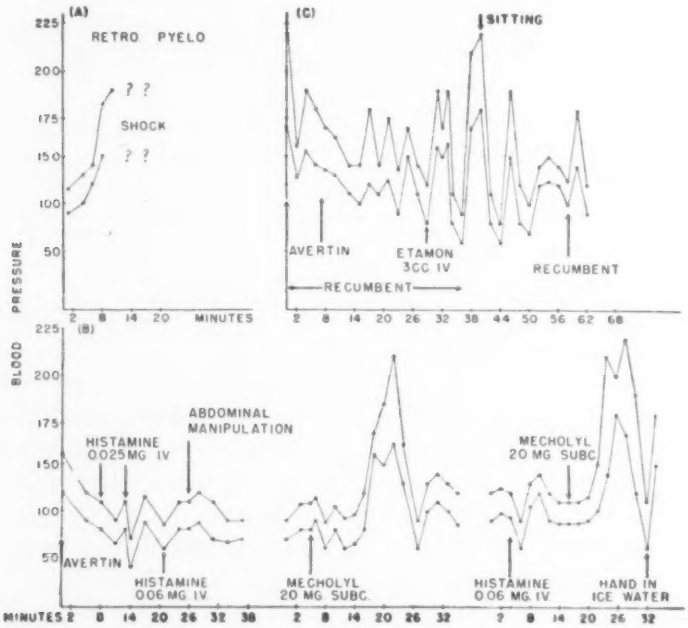
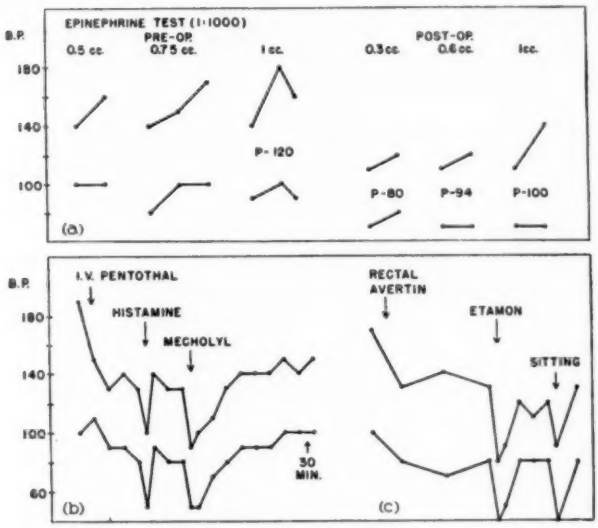


FIG. 5.—(Case 2) Hypertensive reaction at the time of pyelogram (a), and response to specific tests under anesthesia (b and c).

effect on the blood pressure. Since even induction of anesthesia may precipitate an hypertensive reaction, an intravenous needle should be inserted previous to anesthesia to permit rapid administration of whatever medication may be required. Only small amounts of intravenous fluid should be given and blood should be used only to replace that lost if bleeding is encountered during the procedure. Surgical shock is not an important factor and a lowering of the blood pressure, particularly after removal of the tumor, may be guarded against solely by lowering the head in the Trendelenburg position. Actually, death may result from a vigorous attempt to treat this condition as shock, with intravenous fluid and blood and cortical extract, by adding to the burden of the heart in beginning failure. In light of the present knowledge, only two measures are needed: first, an adrenolytic drug to combat the hyper-

FIG. 6



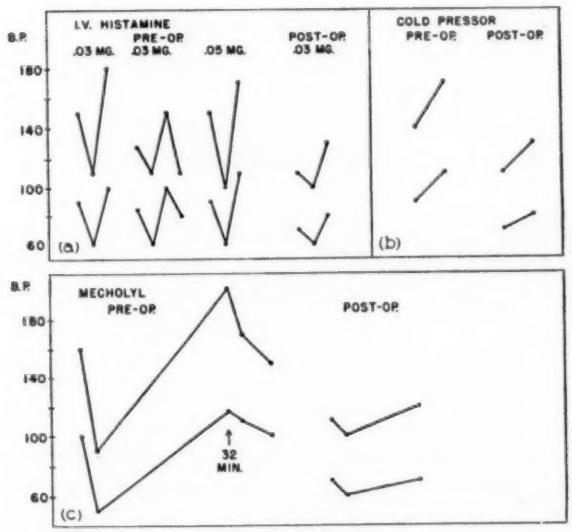


FIG. 7

FIG. 6.—(Case 3) Response: (a) to epinephrine before and after operation; (b) to histamine and mecholyl when patient is under intravenous pentothal, and (c) to etamon while under avertin anesthesia.

FIG. 7.—(Case 3) Response of blood pressure: (a) to histamine before and after operation; (b) to cold pressor tests before and after operation, and (c) to mecholyl before and after operation.

tensive reaction and second, Trendelenburg position to avoid cerebral anoxemia associated with the hypotension following removal of the tumor.

The operation is usually best carried out through the usual kidney incision. In the presence of a large tumor, better access is gained by removal of the twelfth rib, taking care to avoid opening the pleura. The kidney is displaced

FIG. 8

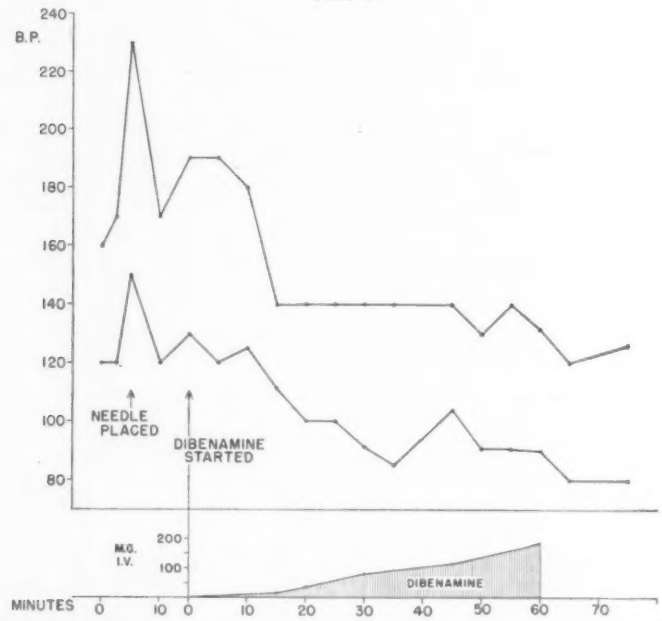


FIG. 9

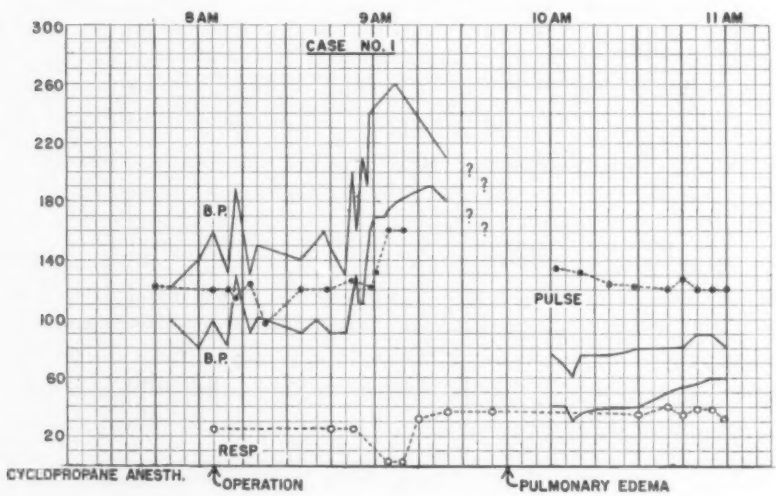


FIG. 8.—(Case 4) Response of blood pressure to intravenous dibenamine.
FIG. 9.—(Case 1) Hypertensive reaction during course of removal of pheochromocytoma with pulmonary edema indicative of acute heart failure.

downward and medially, following which the fascia is incised to expose the adrenal. The identification of the adrenal is facilitated by avoiding exposure of the kidney.

The abdominal approach may be used for large tumors, particularly on the left side or in those cases in which general exploration is indicated, as in Case 2. There seems no reason to employ a thoraco-abdominal incision.

We believe it advisable to explore both adrenal glands, since in 10 per cent of cases the tumors may be multiple and, rarely, there may be congenital absence or atrophy of the other gland. In Cases 1 and 3 the side on which the tumor was located was determined by radiographic visualization before operation, but we explored the opposite adrenal before removing the tumor. It should be recognized that with a hyperfunctioning medullary tumor the opposite adrenal will not show atrophy, contrary to the findings in hyperfunctioning cortical tumors in which atrophy is observed on the opposite side. In Case 4, the side on which the tumor was situated was explored and, before its removal, the opposite adrenal was palpated by opening the peritoneum through the kidney incision. It must be appreciated that this method of exploration may be unsatisfactory. The tumor in Case 2 was removed through an abdominal approach because of a known ovarian tumor. The approach to the left adrenal is similar to that to the tail of the pancreas through the gastrocolic omentum.

In the delivery of the tumor compression should be avoided. The vessel pedicles are dissected and clamped as early as possible to avoid the release of excessive amounts of epinephrine, as noted in Case 1. The amount of adrenolytic drug found during diagnostic studies to be necessary to lower the blood pressure or to prevent the hypertensive reaction can be given intravenously before dissection of the tumor.

Partial or total adrenalectomy should be carried out depending on the exploratory findings. It may be possible to save appreciable portions of the involved adrenal in carrying out the resection of the tumor. If invasive characteristics are noted, the extent of the operation should be determined by the findings, and probably should include nephrectomy. These tumors may be malignant, as in Case 1, in which metastases were found in the cervical nodes removed by radical neck dissection.

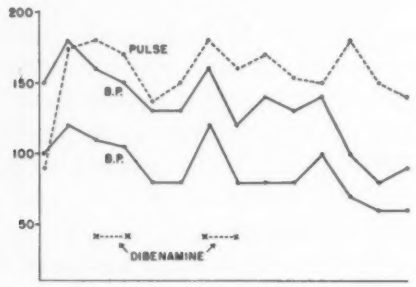
The dramatic and fearsome experiences with the removal of pheochromocytomas, as noted in Case 1, need no longer occur since adrenolytic drugs are now available to avoid the hypertensive reactions. This, combined with the knowledge that the hypotension that continues for a few hours following removal of the tumor need not be feared, removes the unusual danger formerly associated with these operations.

BLOOD PRESSURE RESPONSE DURING AND AFTER OPERATION

In the past great emphasis has been placed on the hypotensive state and collapse with death which may follow removal of a pheochromocytoma. This has been wrongly considered as a state of epinephrine deficiency and injudi-

ciously treated with intravenous epinephrine. Since this collapse state may occur when the pheochromocytoma is still *in situ*, it is evident that epinephrine lack is not the cause. The clinical picture is readily explained on the basis of acute left heart failure resulting from excessive circulating epinephrine, which causes extreme, increased peripheral resistance comparable with that obtained when a surgical clamp is placed on the ascending aorta. One of us[3]

FIG. 10



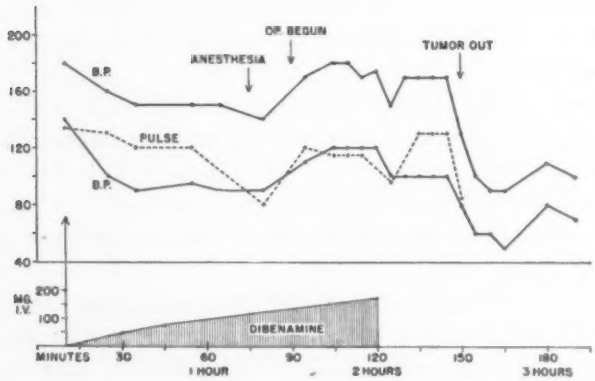


FIG. 11

FIG. 10.—(Case 3) Response of blood pressure and pulse to dibenamine during course of operation.

FIG. 11.—(Case 4) Response of blood pressure and pulse to dibenamine given before and during the course of operation for removal of a pheochromocytoma.

(E. C. B.) first called attention to the cardiac factor responsible for this collapse state. Treating such a state with epinephrine evidences a lack of understanding as to its true cause. A severe hypertensive reaction occurred during the operation in Case 1 (Fig. 9). In Case 2 the operative course was satisfactory. Acute cardiac failure can only be prevented by taking steps to avoid prolonged hypertension during the course of operation. Of more importance is the need of some adrenolytic compound to neutralize the excess circulating epinephrine as reported by Grimson, *et al.*[6] We must, therefore, quickly experiment with the use of benzodioxan or dibenamine during the

course of operation to remove a pheochromocytoma in an effort to find a means of maintaining the blood pressure below a critical hypertensive level.

In Case 3 (Fig. 10) intravenous dibenamine was used during the course of the operation in an attempt to keep the systolic blood pressure below 150. Two short periods of intravenous administration of dibenamine were given, the total dose being 60 mg. Since the pulse remained high there is some doubt as to whether an actual therapeutic dose was given. A hypotensive reaction did not occur after operation.

In Case 4 (Fig. 11), intravenous dibenamine was first given preoperatively to restore the blood pressure and pulse to normal. During the operation dibenamine was continued, giving a total dose of 175 mg., this being the amount which was safely administered during the diagnostic dibenamine test (Fig. 8). The blood pressure was purposely not completely restored to normal, since it was feared a possible hypotensive state might occur if the amount of dibenamine given was in excess. On removal of the tumor the blood pressure fell to 90 systolic and 45 diastolic and then gradually rose to a normal level. Further experience with these new drugs will be needed before their proper use is determined. However, they do give promise of solving the hypertensive reaction during operation to avoid serious postoperative vascular collapse.

	CASES 1	2	3	4
A. LOCATION OF TUMOR SIZE	L 250 GM.	L 4 CM.	R 75 GM.	L 6 CM.
B. B.P. NORMAL	+	+	+	+
C. NO FURTHER ATTACK	+	+	+	
D. B.M.R. NORMAL			+	+
E. GLUCOSE T. TEST NORMAL			+	+
TESTS:				
HISTAMINE		−	−	
MECHOLYL	−	−	−	

FIG. 12.—(Cases 1 to 4) Location, weight or size of tumor and the results of postoperative clinical tests.

Our four patients recovered from the surgical removal of the pheochromocytoma. Only Case 2 caused us concern because of a mild cerebrovascular accident with resulting slight word aphasia which was noted during the postoperative period. In the other patients the postoperative courses were without incident. The blood pressure reached a normal level in those patients with paroxysmal attacks and remained lower than at any time during the preoperative study period (Fig. 12). No further attacks occurred in any of the patients. In Cases 3 and 4 in which the basal metabolic rates were studied they were normal (Fig. 2). In Case 3 the basal metabolic rate had returned practically to normal when determined immediately after removal of the tumor while the patient was still under anesthesia. In Cases 3 and 4 the glucose tolerance tests were normal when taken only a few days after operation (Fig. 3). Histamine tests were done postoperatively in Cases 3 and 4, with normal results. The mecholyl test was also normal after operation in Cases 1, 2 and 3. The hypertensive disease was decisively cured in all four cases. Only Case 1 remains in serious trouble, suffering from metastatic disease. In spite of the malignant growth, no further hypertensive attacks have occurred. Case 2 has residual sight difficulty as a result of serious vascular retinopathy.

SUMMARY

A suspicion of pheochromocytoma is justified in any patient with vasomotor attacks who has such associated symptoms as excess sweating with blotchiness of the skin, toxemia during the early months of pregnancy, findings of fluctuant or sustained hypertension, in whom a suspicion of hyperthyroidism is present.

The salient laboratory findings aiding in the diagnosis of pheochromocytoma are elevation of the metabolic rate without goiter, abnormal pyelograms such as displaced kidney or calcification and elevated blood sugar or a diabetic glucose tolerance test.

Certain incitatory substances such as histamine, mecholyl or etamon are helpful in making the diagnosis of pheochromocytoma. These tests, however, are not specific and must be carefully carried out for proper interpretation. Dibenamine and benzodioxan are also helpful in those patients with pheochromocytoma who have sustained hypertension but sufficient experience has not proved their safety.

The chief danger from surgical removal of pheochromocytoma is a severe hypertensive reaction with subsequent acute left heart failure. This danger can probably be avoided by using an adrenolytic agent during the operation. More experience is necessary before these drugs can be intelligently utilized.

Removal of the tumor leads to cure unless the tumor (pheochromocytoma) is malignant and leads to metastatic disease or has already produced serious vascular damage.

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THE SURGICAL TREATMENT OF HYPERPARATHYROIDISM

WITH A REPORT OF 27 CASES*

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THE SURGICAL TREATMENT of hyperparathyroidism has developed over a period of 35 years. However, before the suggested removal of adenoma of a parathyroid gland by Schlagenhaufer[1] in 1915 and the actual performance of the operation by Felix Mandl[2] in 1925,† many anatomical, physiologic, clinical and pathologic studies had been made in Europe, dating from 1880 when Ivar Sandstrom[3] of Sweden announced the discovery of these minute glands, and including the physiologic studies of Gley[4] in 1890, which differentiated their function from that of the thyroid gland. There were also the clinical and pathologic observations reported by von Recklinghausen[5] (1891), Erdheim[6] (1903), and Askanazy[7] (1904), by whom metabolic disturbance in bone was first associated with a tumor of a parathyroid gland and disturbance of calcium metabolism. Thus, the first case to be correctly diagnosed as one of hyperparathyroidism associated with osteitis fibrosa cystica generalisata was reported by Mandl[2] in Europe in 1925.

In approaching the problem from the standpoint of experimental physiology it remained for Dubois[8] to make the first diagnosis of hyperparathyroidism in America. Knowing of parathormone, which was isolated by Collip[9] and

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949. Since the presentation of this paper the author has operated upon another case in which the patient had bilateral parathyroid tumors, each weighing 15 Gm. The tumors were located behind the right and left inferior poles of the thyroid gland.

† It is interesting that Dr. Charles H. Richardson, of Macon, Georgia, furnished me with this case report of a patient on whom he had operated one year before Mandl's operation. Unfortunately, Doctor Richardson did not publish this operation and the pathologic findings, but the case was thought to be of sufficient interest to be included in this report, if for no other than historical reasons.

"Miss O. M. G., age 25, presented herself in August, 1924, complaining of a lump in the right side of her neck. She gave a history of multiple fractures of the short and long bones. She actually had a kyphosis due to multiple fractures of the vertebrae.

"It appeared on examination that the tumor in her neck was an adenoma of the thyroid gland, because it grew out of the middle of the right lobe of the gland. She was advised to have it removed, with no thought that it might have any bearing on the fractures which she had sustained, and on August 25, 1924, she was submitted to operation. I might add that a basal metabolism test had been normal and there was no evidence of toxicity.

"After the removal of the adenoma, which was about the size of a pigeon egg, it was submitted to the pathologic laboratory, and much to my surprise the report was a parathyroid adenoma.

"We were not doing calcium determinations of the blood at that time, and the case made very little impression except for the fact that it was noted that she had no further fractures. There, you might say, the whole story was dumped in my lap and I missed it.

"She was followed for a number of years and then passed out of sight. Last year I heard that she had died, and on investigation found that she had *died of hypertension and renal failure.*"

Hanson[10] simultaneously, and its effect in producing hypercalcemia in experimental animals, Dubois recognized the disturbance in calcium metabolism in the now famous case of Martel by taking a sample of blood for calcium determination. At a later operation by Churchill[11] in Boston, this same patient was found to have an adenoma of a parathyroid gland in the mediastinum. Great impetus was given by Dubois' report to the study of hyperparathyroidism, and to Albright,[12] Aub and their co-workers, as well as to Collip,[9] must go the credit for establishing the diagnosis of hyperparathyroidism as a notable achievement of the laboratories of experimental and clinical medicine.

With few exceptions, as a result of the determination of hypercalcemia, hypophosphatemia, hypercalciuria and hyperphosphaturia, the diagnosis of hyperparathyroidism can be established with certainty.

Hypercalcemia is observed in hyperparathyroidism; intoxication with vitamin D, as shown by Howard;[13] mutiple myeloma; sarcoidosis; rapid skeletal rarefaction, especially in the young with such conditions as poliomyelitis, and after fracture; osteolytic carcinomatosis; and in some instances of carcinoma of the breast and lung even without roentgen ray or postmortem evidence of skeletal metastases. The major problem in differential diagnosis lies in distinguishing hyperparathyroidism, which is amenable to surgical therapy, from the other conditions. Since the site of parathyroid adenomata is often difficult to ascertain, one would prefer to have positive assurance of the existence of hyperparathyroidism before undertaking the extensive exploration sometimes required to locate the tumor.

Typical uncomplicated hyperparathyroidism, the diagnosis of which is essentially a laboratory procedure, exhibits high serum calcium, low serum phosphorus and high urinary calcium. Albright,[14] however, pointed out that renal insufficiency often accompanies hyperparathyroidism, and that the restriction of phosphorus excretion so imposed may result in normal or even elevated serum phosphorus. Since hypercalcemia *per se* is thought to cause renal damage, and since hypercalciuria with concomitant increased incidence of urinary lithiasis accompanies all types of hypercalcemia, a reason for the high incidence of renal insufficiency and normal or elevated serum phosphorus in all hypercalcemic states is apparent. Furthermore, instances are met in hypercalcemic states other than hyperparathyroidism in which the serum phosphorus is low. (Howard[13])

Thus there are times when the chemical diagnostic criteria of hyperparathyroidism are not distinctive. Collateral evidence such as the history, physical examination, and blood morphology (anemia is common in all the diseased states in which we have hypercalcemia) may not yield the information necessary for diagnosis. Skeletal and pulmonary roentgenography may not disclose sarcoid or metastasis; patients with myeloma and sarcoid may have hypercalcemia either with or without hyperproteinemia, and myeloma may be accompanied by general skeletal rarefaction without characteristic punched-out lesions.

Soon after Collip's[9] isolation of the active principle of the parathyroid glands the observation was made and confirmed that there is little or no reflection in the cerebrospinal fluid of the high and low serum calcium seen in the hyper- and hypoparathyroid states. It occurred to Howard[15] that perhaps in hypercalcemic states other than hyperparathyroidism, the calcium of the cerebrospinal fluid might be elevated, and that this might serve as a distinguishing feature useful to the clinician. The normal variation of cerebrospinal fluid calcium is between 4.5 and 5.5 mg. per 100 cc. It was found by Howard[15] and others that the spinal fluid calcium in all patients with hypercalcemia other than that seen in hyperparathyroidism was elevated. In four patients with hyperparathyroidism in the series of 27 to be reported, the cerebrospinal fluid calcium was lower than the calculated diffusible calcium in the serum, and in only one patient was the cerebrospinal fluid calcium elevated to 6 mg. per 100 cc. Thus it would appear that in hypercalcemic states, at least those with normal total serum protein, elevation of the calcium of the cerebrospinal fluid above 6 mg. may be interpreted as presumptive evidence against hyperparathyroidism. There seems to be a cerebrospinal block which behaves differently towards the hypercalcemia of hyperparathyroidism than towards the hypercalcemia produced by other diseased states. Opinion is still lacking as to how calcium is "carried" into the plasma, yet it seems of considerable interest that the rest of the body tissues respond in a seemingly similar manner to hypercalcemia however induced, but the cerebrospinal fluid block does not.

It is now recognized that the surgeon must accept the fact that adenoma or primary hyperplasia of the parathyroid glands exists in every case in which these changes in the cerebrospinal fluid, blood, and urine have been demonstrated and must therefore be prepared to prolong the dissection until such abnormal parathyroid tissue is found. The surgical problems associated with hyperparathyroidism broadly considered are primarily concerned with the finding and removal of the parathyroid tumors and the recognition, with subtotal resection, of hyperplastic tissue in cases of diffuse primary hyperplasia. The difficulty of recognition of the parathyroid glands and the many and varied locations in which they may normally occur complicate the operation. Furthermore, variation in size and number of tumors of the parathyroid glands as well as the differentiation of adenoma from diffuse hyperplasia may present a very complicated proposition for the surgeon.

In 1931 Walton[16] reported in the British Journal of Surgery operations upon four patients with hyperparathyroidism due to adenomata of the parathyroid glands, and for the first time called attention to the necessity of seeking for abnormal parathyroid tissue elsewhere than in the traditionally described regions about the thyroid gland. Walton's observation was based on his experience in operating upon two patients with adenomata of parathyroid glands which had been displaced mechanically from their original positions near the thyroid gland. He called attention to the fact that if there is such displacement one may determine the type of displacement by the preservation

of the vascular pedicle connecting the tumor by blood supply to the original thyroid artery (Fig. 1).

In 1934 and 1936 Churchill[11] and Cope[17] reported 11 patients with adenomata of the parathyroid glands who had been operated upon at the Massachusetts General Hospital, and for the first time correlated the displacement of adenomata of the parathyroid tissue with the embryogenesis of the parathyroid glands. In order to explain the unusual locations in which normal and also abnormal parathyroid tissue may be found throughout the paths of migration of superior and inferior parathyroid glands in the embryo and

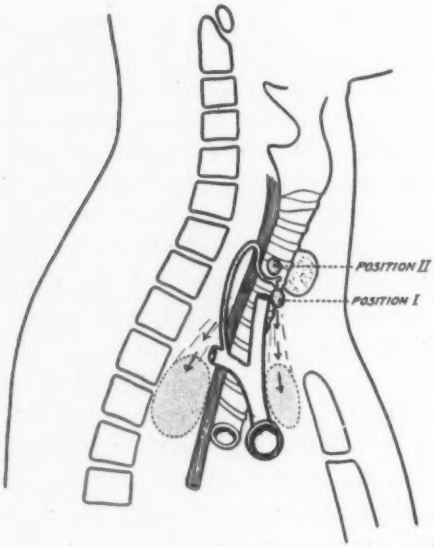

FIG. 1

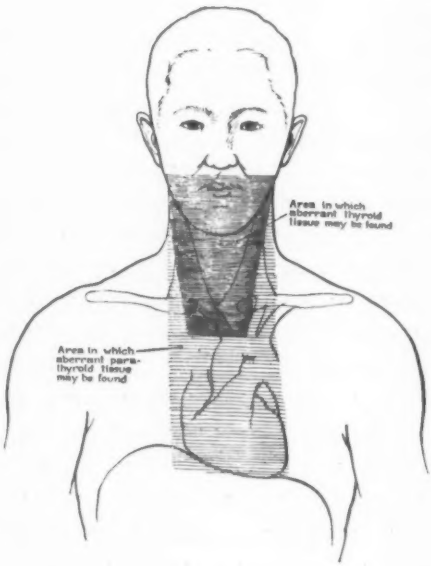

FIG. 2

FIG. 1.—Photograph from Figure 206 in Walton's article, showing possible course of mechanically displaced adenomata of parathyroid gland. If enlargement occurs in position 2, that is, posterior to the thyroid gland, the adenoma will be displaced in the posterior superior mediastinum. If the parathyroid gland occupied position 1, adenoma developing in this position ventral to the great vessels will be displaced in the anterior superior mediastinum. Vascular pedicles will be attached.

FIG. 2.—Diagrammatic sketch made by author to demonstrate fascial tube running from cervical region to mediastinum.

hence later in the adult, Churchill[11] further called attention to the fact that the parathyroid glands, and therefore any abnormal parathyroid tissue, would probably be found within the bounds of the tubular fascial structure of the neck bounded anteriorly by the deep layer of the middle cervical fascia and posteriorly by the prevertebral fascia (Fig. 2). These observations by Walton, Churchill and Cope led to the systematic surgical dissection of the cervical region as well as the posterior superior and anterior superior mediastinum, and for the first time placed the operation for adenomata of the parathyroid glands not only on an anatomical but an embryologic basis.

In 1937 Norris[18] published his excellent article on the embryologic development of the parathyroid glands in man, in which the embryologic pathways of the parathyroid glands were conclusively demonstrated and their relation to the lateral thyroid body from the fourth branchial cleft, as well as to the thymus from the third branchial cleft, was shown.

Cope,[19] in 1941, correlated the clinical experience of the patients operated upon for adenomata of the parathyroid glands with the possible embryologic pathways described by Norris.[18] He called special attention to the fact that tumors may develop elsewhere than in the normal locations of the parathyroid glands in the neck and mediastinum, and again drew attention to the displacement of the parathyroids due to mechanical dislocation. In other words, abnormal parathyroid tissue can develop in a parathyroid gland in the vicinity of the thyroid gland, or in parathyroid glands which have been carried into the mediastinum during their embryologic development, and also in normally located parathyroid glands which, because of gravitation or thoracic aspiration, become dislocated into the anterior or posterior mediastinum, in which event the abnormal parathyroid tissue will have a vascular pedicle arising from the parent thyroid artery. It was also noted by Cope[19] that abnormal parathyroid tissue developing in the embryologically displaced normal parathyroid glands in the anterior mediastinum, in the areolar tissue and in the thymus gland, as a rule receives its blood supply locally from pleural vessels in the immediate vicinity of the adenoma. The same statement may be made regarding the author's two cases of intrathyroid adenomata.

In January, 1947, Norris[20] reported a study of 322 cases of hyperparathyroidism which covered the years from 1903 to 1946. In 1948 Black[21] reported an additional 63 patients from the Mayo Clinic. In recent personal communications Cope stated that 104 cases of hyperparathyroidism have been treated up to the present time at the Massachusetts General Hospital. Black listed 106 cases from the Mayo Clinic; Lahey listed 31 cases unreported from the Lahey Clinic; and seven unreported cases from the Ford Hospital are listed by McClure. These, in addition to the 27 cases included in this report, bring the sum total of instances of hyperparathyroidism that have been reported since 1903, or will be reported in the near future, to 597. Undoubtedly there are many other cases throughout the country that have not appeared in the literature, and perhaps there have been reports from other clinics of which I am not aware. The incidence of hyperparathyroidism in this country is, of course, unknown. There are undoubtedly a great many cases, particularly those with renal complications, that remain undiagnosed. The clinical manifestations of hyperparathyroidism, particularly in its early stages, are obscure in their onset and misleading in their progress. Unfortunately, too often the condition is noted only when the disabling and perhaps fatal complications of the disease become evident, and in the past these complications have been mistakenly treated instead of the fundamental underlying cause of the disease.

It has been shown that whenever attention is directed to a particular syndrome the incidence of this condition seems to increase. I think this is brought

out well in Black's[21] report from the Mayo Clinic. From 1928 to 1942 the diagnosis was proved in 12 cases only. After sufficient interest was manifested, in the last four years of his study 47 cases of hyperparathyroidism were diagnosed.

The recognition of the fact that probably the most frequent complication of hyperparathyroidism is some form of renal lithiasis or deposition of calcium at one point or another in the urinary tract has been mainly responsible for the noticeable increase in the diagnosis of this disease in not only the Mayo Clinic but the Johns Hopkins and Massachusetts General Hospitals. The concerted effort to be certain of the calcium phosphorous ratio in all cases of renal lithiasis, particularly those of recurring urinary calculi, has brought to light the fact that the heretofore higher incidence of bony complications over renal complications was probably because the renal complications were unknown or were overlooked by earlier observers.

As emphasized by Lahey,[22] the, as yet meager, follow-up studies would certainly indicate that the main goal for the clinical approach in the treatment of hyperparathyroidism is to make the diagnosis at an earlier stage in the disease before the more serious, deforming and sometimes fatal skeletal and renal complications have occurred. It should be noted that nephrocalcinosis in its various forms may not be the sole cause of the marked renal insufficiency which we all now recognize as an even more frequent and serious complication of hyperparathyroidism than decalcification fibrosis of bone.

It is my opinion that even the relatively small series of 27 cases of hyperparathyroidism which I wish to report should be recorded in order to stimulate further reports from other individuals and clinics so that more material will be available for study. Twenty-five of our cases have been due to adenoma of the parathyroid glands, one to diffuse hypertrophy and hyperplasia of four parathyroid glands, and one to carcinoma of a parathyroid gland which recurred locally, metastasized, and was associated with renal and skeletal complications, together with the typical disturbances of the blood chemical formula. This case has been reported by Skinner[23] and others.

Of the 25 cases of hyperparathyroidism in this series which have been proved to be due to benign tumor or adenoma of the parathyroid glands, in two instances there were two adenomata. In one case the adenomata were discovered at the first operation; in the second case the second adenoma was removed a year after the primary operation for removal of the first tumor, thus making a total of 27 adenomata of the parathyroid glands.

Although this series is far too small for exact statistical conclusions, it is interesting that, contrary to some reports, the sex distribution was essentially even: 14 of the patients being males and 12 females. The age factor was not unusual, the ages of the males varying from 10 to 53, and of the females from 14 to 56. These ages, of course, were noted at the time the treatment was carried out.

In one male patient the cause of the hyperparathyroidism was diffuse hypertrophy and hyperplasia of the four parathyroid glands. Clinically, how-

ever, the syndrome was indistinguishable from the clinical manifestations produced by adenoma of a single gland.

In the entire series there were complications as a result of the long-standing hypercalcinemia and hypophosphatemia, but it was interesting to see that in the male patients 13, or 92.8 per cent, had only renal changes with stones, with no bony changes whatever. In one male, or 7.2 per cent of the men suffering from the disease, there were bony changes which were diagnosed as osteitis fibrosa cystica, with no renal involvement or evidence of any urinary calculi. The percentage of renal complications was far greater in the males than in the females. Six of the 12 women suffering with hyperparathyroidism, or 50 per cent, manifested only skeletal changes without renal complications, whereas the reverse was true of five, or 41.6 per cent, who had renal stones only without any evidence of bony changes. In one female in whom the tumor weighed 58 Gm., the largest one in our series, there were both bony changes and renal stones. Hence it may be said that in this small series of cases the renal complications outnumber the osseous, the males being thus affected far more frequently than the females. As stated before, in two cases (7.7 per cent) there were multiple adenomata, two having been removed at operation in both instances. Two adenomata were buried deeply in the thyroid gland, one small, weighing .5 Gm. and the other 58 Gm. In two instances the adenomata were both visible and palpable. In the patient with the 58 Gm. adenoma, which was a true intrathyroid adenoma, this occupied the left lower lobe of the thyroid, and could be seen and readily palpated. The second patient was a 14-year-old girl, in whom there were two adenomata, one situated posterolateral to the right lobe of the thyroid gland about the junction of the upper and middle thirds of the lobe, the other adenoma occupying a position posterior to the left upper pole. The right tumor could be seen and readily palpated. Of the 25 cases in which there were a total of 27 adenomata, in five (18.6 per cent) the adenomata were found in the mediastinum—three in the anterior, one in the lateral superior and one in the posterior superior region. Twenty-two adenomata (81.4 per cent) were found in the cervical region in the immediate vicinity of the thyroid gland, as shown by the diagram. In one patient in whom two adenomata were found, the second was removed elsewhere one year after his primary operation in the Johns Hopkins Hospital. This tumor, not shown in Fig. 3, lay inferior to the lower pole of the left lobe of the thyroid gland.

An attempt to correlate the size of the tumors, which varied in these cases from .5 Gm. to .58 Gm., with the elevation of blood calcium did not reveal the information that had been anticipated. In the first place, in this series there was on the whole no very great difference in size except in the one instance of the tumor weighing 58 Gm., the remainder of the adenomata ranging from .5 to 6 Gm. It was interesting to compare the elevation of the blood calcium in the patients with renal as well as bony complications. It was found that the mean average elevation of calcium in the renal cases was 15.2 mg. per 100 cc. as compared to 14.4 mg. per 100 cc. for those showing only bony changes, whereas the phosphorus was 2.8 and 2.4 mg. per 100 cc.

respectively. The blood phosphatase in all the patients having renal complications only was within normal limits; that is to say, not above 4 to 5 Bodansky units. On the other hand, in each instance in which there were skeletal complications the serum phosphatase was elevated. However, we are generally in agreement with Castleman and Mallory[24] as well as Cope[19] that there is

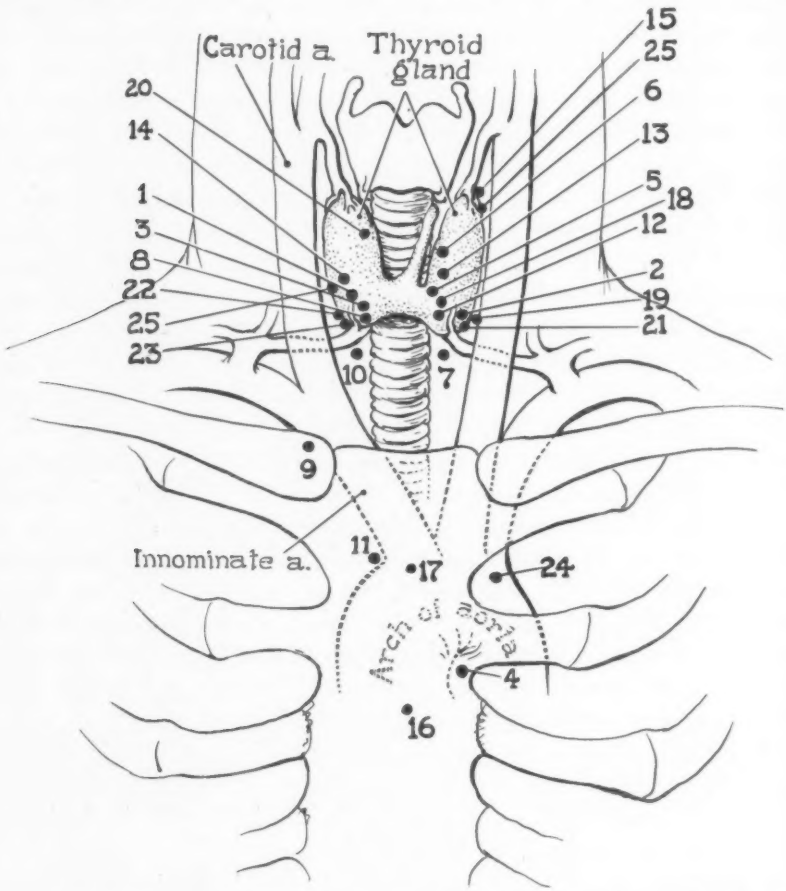

FIG. 3.—Diagrammatic sketch showing location of adenomata of the parathyroid in 25 cases of hyperparathyroidism reported in this series. It will be noted that No. 25 is shown both at the right lower and left upper poles. There was an additional adenoma removed a year later (not shown in this diagram) which lay beneath the left lower pole, making a total of 27 adenomata in 25 cases.

probably some correlation that would be revealed in a larger series between the intensity of the disease as judged by the changed values for calcium and phosphorus in the blood and the size of the tumor. The production of parathormone in the patient with the 58 Gm. adenoma must have been very much reduced; otherwise the patient would have succumbed to acute hyperparathyroidism. As pointed out by Castleman and Mallory,[24] the larger tumors within certain limits probably are biologically less active than the smaller ones.

Following operation there was one death. The patient, who was 53 years of age, had severe tetany, and died of bronchopneumonia four days postoperatively. There were no renal complications, but there was pronounced decalcification of the bones. The adenoma of the parathyroid was very small, measuring between 2 to 3 mm. in its greatest diameter. The weight, unfortunately, was not recorded. The preoperative blood calcium was not greatly elevated, being only 11.3 mg. per 100 cc.; the phosphorus was 3.3 mg. per 100 cc., the total protein 6.9, the serum phosphatase 5 Bodansky units. The patient was operated upon on April 7, 1937. Between that date and the onset of fatal tetany with convulsions, delirium and bronchopneumonia on April 11, the blood calcium was around 8 mg. per 100 cc. and the phosphorus averaged 5 mg. per 100 cc. On April 11, the blood calcium fell to 5.6 mg. per 100 cc. and the phosphorus rose to the extraordinary level of 8.2 mg. per 100 cc. The patient succumbed on April 12. The pre- and postoperative non-protein nitrogen in the blood was within normal limits. There was no clinical evidence of renal damage. The sudden and inexplicable rise in the blood phosphorus was most interesting. However, death occurred in spite of efforts to combat the tetany by the use of parathormone, calcium chloride and calcium gluconate.

There were six other cases of postoperative, rather severe tetany, and in each of these—five females and one male—there were rather extensive skeletal changes with no renal complications. In one additional female there was slight tetany. In this patient, who had renal involvement without bony involvement, there was slight postoperative tetany which required no treatment. This patient was the girl of 14 years, who had two adenomata weighing over 3 Gm. each. It was felt that the tetany developing in the patients with skeletal changes as a result of decalcification was produced by draining of the serum calcium ions into the bony reservoir after an excessive secretion of parathormone had been removed following resection of the adenoma, whereas in the young girl with two adenomata and no bony involvement the slight tetany was attributed to the temporary hypofunction of the remaining atrophic right superior and left inferior normal parathyroid glands. Other than the instance referred to above there was no postoperative tetany in any of the patients in whom only renal complications were present, in contradistinction to those in whom there were changes in the osseous skeleton. In all the patients in whom tetany developed following operation, excepting the above mentioned fatal case, the signs and symptoms disappeared promptly following oral administration of calcium chloride.

In six of the patients in this series there was so-called metastatic calcification. In one female and one male, following renal involvement only, there were crystals in the conjunctivae. In two males with renal involvement only there were pancreatic calculi associated with diabetes (Fig. 4). In one male with complication of the disease limited to the skeleton, there was calcification of the aorta and calcified areas in the lung. In another male in whom the

complication was limited to the kidney, there was extreme calcification of the vessels of the leg.

In this series there was one postoperative death, or a mortality of 3.8 per cent, and one recurrence of an adenoma.

Although some pertinent follow-up data are lacking in this relatively small group of cases, the data may still prove to be somewhat instructive as to prognosis if not as to successful therapeutic measures. In the 25 cases in which hyperparathyroidism was proved to be due to adenomata of the parathyroid glands, there have been nine deaths (four females and five males) following discharge from the hospital, after periods of time ranging from three to 11 years. All of the deaths have been due to hypertension with or without renal insufficiency. All of the patients except one female, age 25 years, were in their fourth or fifth decade at the time of death. In five of the nine the fatal hypertension occurred in patients who at the time of their hospitalization had no renal, but only skeletal complications. In one patient there were both renal and skeletal complications. In the remaining four there were no bony complications, but only renal complications. Thus it can be stated from this meager follow-up study that, although at the time of the operation for removal of the offending adenoma, the patients appeared well of their hyperparathyroidism as far as one could judge from the return of their blood calcium and phosphorus and phosphatase to normal, nevertheless there was a continuous degenerating process going on for a number of years that produced complications resulting in renal insufficiency and hypertension in some, and in others in hypertension without renal complications. Obviously, forces were freed during the course of the hyperparathyroidism which could not be checked after the deleterious effects of the excessive secretion of parathormone had been stopped. Thus, in 25 cases in which the hyperparathyroidism was due to adenomata of the parathyroid glands there was an immediate mor-

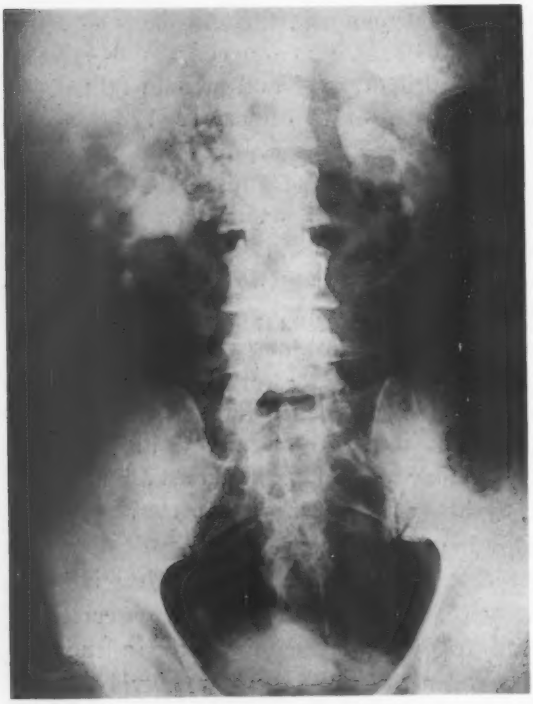

FIG. 4.—Demonstrating metastatic calcification in a male patient with renal lithiasis; metastatic calcification in the pancreas where multiple pancreatic stones can be observed.

tality of one, or 4 per cent, with an ultimate mortality of 35 per cent to date. It is interesting that the one male patient with diffuse hypertrophy and hyperplasia of all four parathyroid glands had no renal or skeletal complications which were demonstrable clinically, the preoperative nonprotein nitrogen averaged 60, and the postoperative blood calcium and phosphorus returned to normal and remained so until the time of his death seven years later of uremia, hypertension and anemia. It is interesting that this patient, in spite of no demonstrable evidence, must have had renal damage during and after his phase of hyperparathyroidism.

On one patient there was insufficient information to arrive at any conclusions as to his present state of health. Of those living and apparently well, in only one female with definite skeletal complications was reossification complete within a year. In this group of 14 patients there were seven females and seven males.

As demonstrated by the clinical analysis of the 14 male patients at the time of operation, the only male with skeletal complications did not survive, whereas seven of the 13 with renal complications survived. Four females with renal complications alone are still living, and three females who had skeletal complications without renal involvement are still alive, although not all are completely free of symptoms. One female who had only skeletal complications, has a great deal of discomfort three years after operation, which probably originates in her skeleton, for she complains of being miserable and having a great deal of pain, which necessitates frequent hospitalization. Another instance, a male of 40 years, with renal complications only, who has some postoperative hypertension with definite damage to the kidney is far from well, as his hypertension is progressive and undoubtedly will end fatally before many years. A third instance, a female of 66 years, with only skeletal complications, is well as far as hyperparathyroidism and the level of her blood calcium and phosphorus are concerned; nevertheless she suffers from severe pain and discomfort that is supposed to have its origin in her skeleton. Four of the patients have had recurring attacks of renal colic and formation of stones, although their blood chemistry indicates that parathyroidism is no longer present. Thus, although 14 patients, or 56 per cent, are alive, it would be misleading and incorrect to state that they are perfectly well. As stated above, we were unable to obtain any information about one patient, and it is not known whether he is still alive.

If we can draw any conclusions from such a small group of patients, it is that the individual in whom there is the least evidence of renal damage, and in whom the preoperative blood pressure and nonprotein nitrogen and the blood calcium and serum phosphorus return to normal levels within a reasonable time postoperatively, has the best outlook for complete recovery. Once complications have developed to any extent, particularly in the fourth and fifth decades, in spite of the successful removal of abnormal parathyroid tissue and a complete cure of the hyperparathyroidism as evidenced by a return to normal of the blood calcium and phosphorus and blood phosphatase, the sec-

ondary damage to the kidney will not be corrected. Apparently there is a progressive renal insufficiency which has not as yet been completely explained but which probably has to do with the retention or excretion or both of phosphorus. The study emphasizes the point mentioned by Lahey,[22] however, that our main goal in therapy should be to attempt to make the diagnosis of hyperparathyroidism before obvious and serious complications have occurred.

NORMAL PARATHYROID GLAND

In order that the surgeon dealing with hyperparathyroidism may be as precise and systematic as possible in the surgical dissection, the recognition of the normal parathyroid glands grossly is of the greatest importance. The ability to recognize these minute glands can be attained only by having first-hand knowledge of the variations in their size, number, color and location.

A normal parathyroid gland weighs 30 to 40 mg. and measures 6 to 7 by 3 to 4 by 1.5 to 2 mm. Welsh[25] reported the largest parathyroid gland, measuring 15 by 6 by 3 mm., and the heaviest, weighing 100 mg. If two parathyroid glands should be fused on one side, the normal glands may approach the size of a parathyroid tumor. The most frequent locations of the parathyroid bodies are shown in Fig. 5 which is reproduced from the diagram made by Dr. William G. MacCallum[26] following 67 dissections of the parathyroid glands at autopsy. The number of glands may vary from two to six or even more. Gilmour and Martin,[27] in a study of the parathyroid glands of 527 persons on whom autopsy was performed, found four glands in approximately 80 per cent, more than four in 6 per cent, and fewer than four in 14 per cent. In some instances supernumerary glands were found bringing the total number to five or six glands in an individual; where only two or three glands have been demonstrated one suspects that normal parathyroid tissue may have been displaced and not revealed by the dissection. Let it suffice to say that in the great majority of individuals there are generally four parathyroid glands.

The size and shape of the normal parathyroid gland shows considerable variation depending on the location and the structures in the immediate vicinity. The composition of the gland consists mainly of parathyroid cells and fat cells, with but little supporting stroma. The capsule of the gland is extremely thin, with a fine weblike network of capillary vessels over it. Because of its soft consistency, the shape of the parathyroid gland is largely determined by the surrounding organs or structures. It may be flat if it is molded over the surface of the trachea, thyroid or esophagus; when free in the surrounding fatty areolar tissue it may be almost globular in shape. The vascular hilus from which develops the pattern of blood vessels over the surface of the gland is a great aid in the identification of these structures.

The color of the normal gland varies according to the fat content. Before puberty the gland is composed of chief cells and has a characteristic coffee-brown color. Fat cells which dilute the brown color with varying admixtures of yellow appear at puberty, increasing progressively until the person attains

the age of about 40 years. During middle age the fatty tissue remains fairly constant. After the age of 40 there may be a decrease in the fat content, with almost none present in individuals of the older age groups.

Identification of the normal parathyroid gland is a most important step in the systematic dissection for the exposure of abnormal parathyroid tissue, because in all probability an atrophy of disuse of the gland cells, with relative increase in fat cells, makes identification of the normal parathyroid glandules in the presence of the active tumor extremely difficult. Since fat cells are absent in the adenomatous tissue as well as in the hyperplastic glands the color is darker brown than that of the normal adult gland.

The most common location of normal as well as abnormal parathyroid tissue is in the region of the thyroid gland, as shown by the diagram of MacCallum[26] (Fig. 5). The abnormal location of many parathyroid glands is dependent in part on the location of their primordia during the embryologic descent in association with lateral thyroid bodies or thymus or both, and in part on the displacement of the normal gland, particularly if the latter is enlarged by both gravity and negative intrathoracic pressure. Thus, the surgeon who attempts to operate for the treatment of hyperparathyroidism must have accurate knowledge of the normal parathyroids and their locations as well as the embryogenesis and the possibilities of mechanical displacement. That there are both normal and abnormal locations constitutes the fundamental anatomical basis for the surgery of hyperparathyroidism.

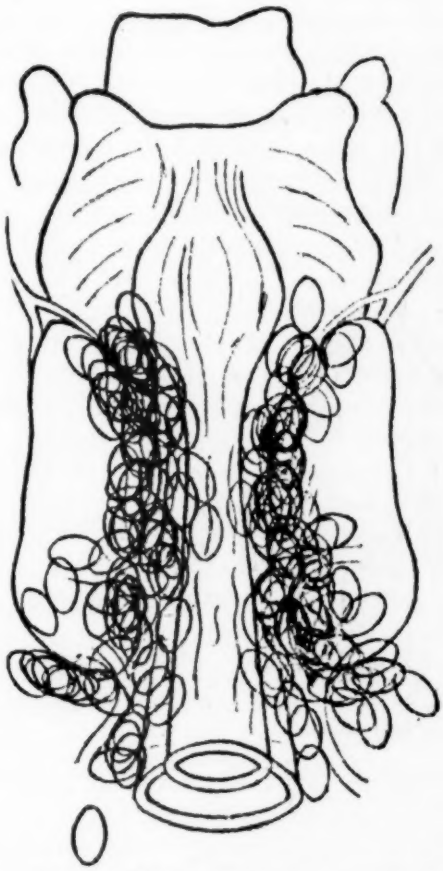

FIG. 5.—Diagrammatic sketch made by Dr. Wm. G. MacCallum to demonstrate the usual location of the parathyroid glands found in 67 autopsies. The outlines of the glandules which are most heavily marked are the sites of the usual location. The numerous variations from this usual location can be readily seen.

A brief reference may be made here, for the sake of emphasis, to the embryogenesis of the superior and inferior parathyroid glands. Whereas it is true that at least 80 per cent of the parathyroid tissue, both normal and abnormal, is found in the region of the thyroid gland, the remaining 20 per

cent may be displaced either because of the embryologic descent of this tissue or the mechanical displacement of an enlarged gland. The parathyroid glands develop as two pairs from two separate bilateral primordia in conjunction with the thyroid and thymus glands (Fig. 6). The superior parathyroid glands develop in close association with the lateral anlage of the thyroid gland (Fig. 6). The development of the upper pair of parathyroid glands is the simplest; arising from the fourth branchial cleft, the lateral parathyroid primordia appear above and behind the lateral thyroid component arising from the same cleft and descend in this relation to the lateral thyroid body as it descends into the neck to join the median thyroid component. On the basis of the embryologic migration, the superior parathyroid glands should lie between the fascial tube, bounded anteriorly by a deep layer of middle cervical fascia and posteriorly by the prevertebral fascia (Fig. 2). The superior glands, although having their origin from the fourth cleft in the embryo, are so limited in their descent that in the adult they become the superior parathyroid. The glands are situated well posteriorly and lie on the pharynx and esophagus, but can lie on the thyroid gland posteriorly or even anteriorly. The most common position of the superior parathyroid glands is about at the junction of the middle and upper thirds of the lobe of the thyroid gland. They lie in a plane dorsal to the recurrent laryngeal nerve and inferior thyroid artery. Because of their limited embryologic migration the identification of the superior parathyroid glands is, as a rule, easier than that of the inferior parathyroid glands (Fig. 7). The anlage of the adult inferior parathyroid arises from the third branchial cleft rostral to that of the superior gland and migrates caudally, along with the thymus, lateral and ventral to the pathway of the superior parathyroid and thyroid glands (Fig. 8). Although the inferior parathyroid glands have their origin cephalward, the embryologic pathway, as it descends with the thymus, covers a much longer course from at least 1 cm. above the larynx to any position above the diaphragm in the anterior mediastinum. These embryologic pathways have been beautifully described by Weller[28] and later by Norris,[20] and the correlation of the clinical deposition of parathyroid tissue with the embryologic development was made possible by their fundamental researches.

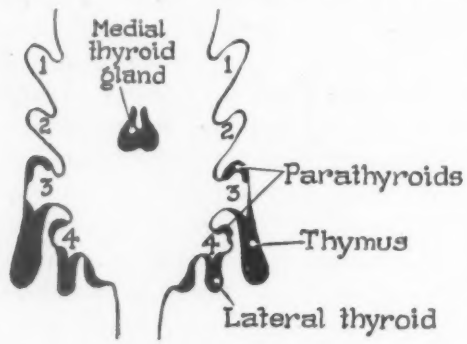

FIG. 6.—Diagrammatic sketch of branchiogenic system demonstrating origin of the parathyroid glands from the third and fourth branchial clefts.

As a rule, in the embryologic development the inferior parathyroid, or Parathyroid III, is deposited opposite the lower pole of the thyroid gland or just below and ventral to the inferior thyroid artery, the recurrent laryngeal nerve, or even the inferior thyroid veins. Sometimes, however, the inferior

parathyroid glands descend beyond the lower pole of the thyroid and are deposited low in the anterior neck and into the anterior mediastinum. Again, the inferior gland may remain within the capsule of the thymus or, in the event that the thymus descends incompletely into the neck, depositing cervical aberrant thymus tissue, the inferior gland may be found in the region of this tissue or beneath the capsule. It is doubtful whether the inferior parathyroid gland ever descends beyond the lowest level of the thymus.

In the adult, the inferior parathyroid glands may also be found in the visceral compartments of the cervical fascia at any level from the upper border of the larynx to and including the anterior mediastinum. As has been pointed out for many years, the most common location of the inferior glands is near the posterolateral surface and inferior poles of the thyroid glands, anterior to the branches of the inferior thyroid artery and recurrent laryngeal nerve. In addition to the variation in position of the superior and inferior parathyroids because of their embryogenesis and the descent along the embryologic pathways, when enlarged they may be displaced caudally through the superior strait of the thorax due to gravity or intrathoracic suction (Fig. 9). It may be seen from these diagrams that if the parathyroid glands, either superior or inferior, in the adult are situated well posteriorly, the displacement is towards or in the posterior superior mediastinum, and if it is situated more anteriorly it would more than likely descend into the anterior superior mediastinum. Because of the dorsal position of the superior parathyroid glands the displacement in the adult person is down or into the posterior mediastinum, and the inferior glands may be displaced into either the anterior or the posterior superior mediastinum, depending on its position before displacement. We have never seen a parathyroid gland on the anterior surface of the thyroid gland, but in two instances the glands were situated within the thyroid.

As mentioned by Walton[16] and emphasized by Cope,[19] enlarged parathyroid glands, or adenomata which have been displaced mechanically, receive their blood supply from the superior and inferior thyroid arteries or from the anastomotic artery joining the former two, which course along the posterior surface of the thyroid lobe. When the gland develops in the mediastinum it may receive its blood supply locally from any neighboring artery.

In the case of mechanical dislocation, the telltale pedicle can usually be detected, and thus the path of departure of the enlarged parathyroid from its normal position may be determined. As pointed out by Black,[21] all parathyroid glands in the posterior superior part of the mediastinum have pedicles, whereas those in the anterior and superior parts may or may not have pedicles. In this series of cases there have been no instances in which normal or abnormal parathyroid tissue has been found within the thymus, but it is well to bear in mind that this location is possible.

Depending on the size of the enlarged parathyroid glands, and in the event that they are the site of adenomata arising from the embryologic displaced parathyroid tissue, those that lie lateral to the thymus may drop into the

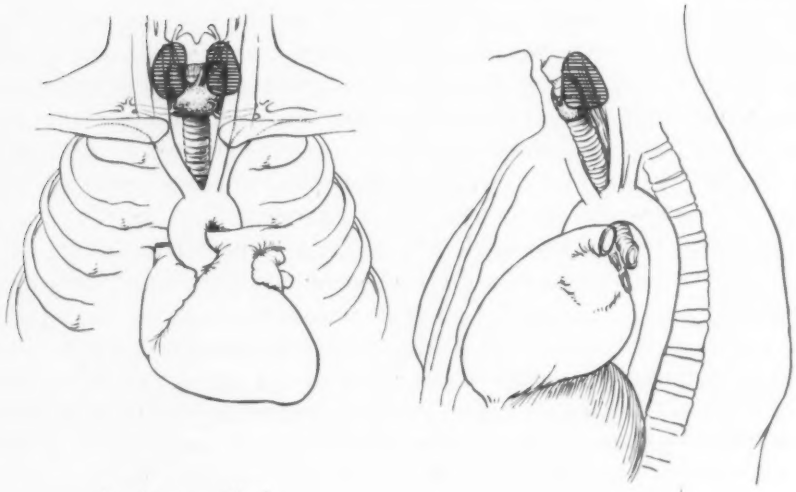

FIG. 7

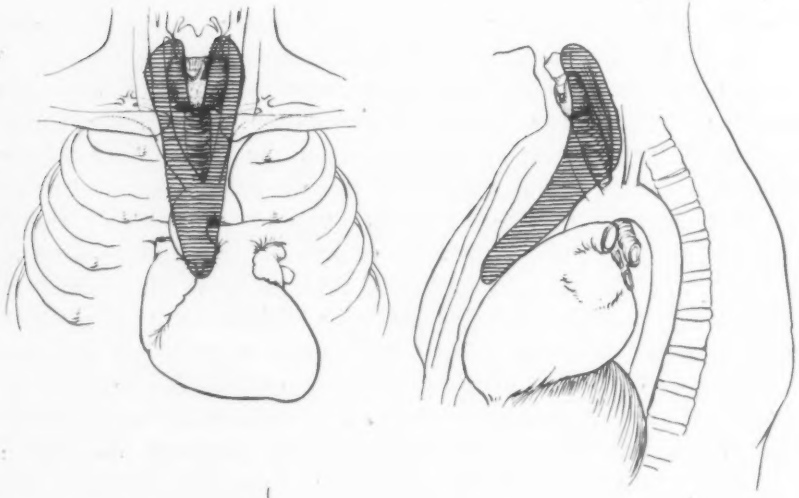

FIG. 8

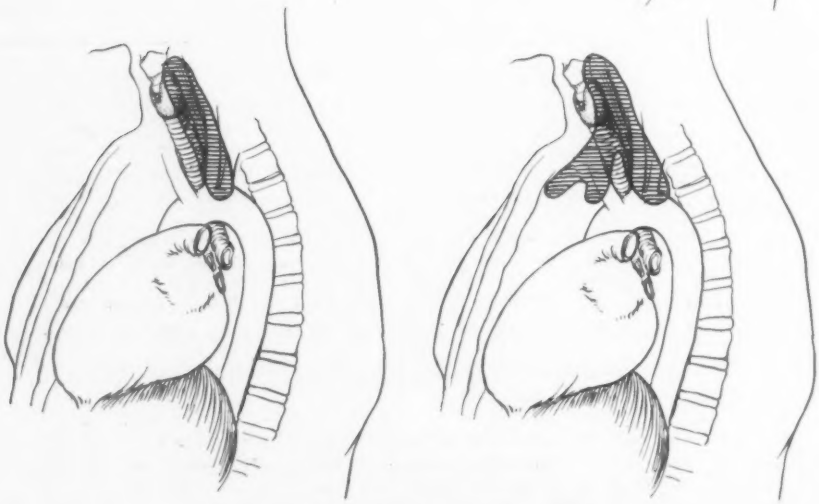

FIG. 9

(Legends on opposite page)

middle posterior mediastinum from the original position in the anterior mediastinum. As shown in Fig. 3, the most common region in which adenomata of the parathyroid have been found in these 25 cases is in the vicinity of the inferior thyroid poles. However, it may be that the adenomata located in this region represent tumors that have developed in the superior glands and descended caudally, and not necessarily, as suggested by Norris,[20] that the inferior parathyroid glands are more susceptible than the superior to the formation of benign tumors. Many more instances of adenomata of the parathyroid will have to be carefully analyzed before this question can be settled.

Previous communications have discussed the variation in location of parathyroid glands. However, since cases are still being encountered in which a systematic dissection based on the above principles with regard to normal and abnormal locations of parathyroid tissue is not carried out, it is felt that these points are worthy of restatement and further emphasis.

Operative Technic. The rationale of the operative procedure and the technic must be based on search primarily of the cervical region and secondarily of the mediastinum. Such a search must be systematic and, as previously emphasized in the literature, the operative field must be dissected carefully without soiling from hemorrhage. After the usual thyroid incision with reflection of the skin flaps and platysma, as well as division of the preglandular muscles has been made, a routine course should be followed. Examination is first made of the right lobe of the thyroid, to note whether or not this lobe is the site of intrathyroid nodules. The right lobe is then mobilized and rotated medially, exposing first the right lower pole, the inferior recurrent laryngeal nerve, and the inferior thyroid artery, as well as the inferior thyroid veins. If the search about the lower pole has been fruitless, the dissection is then carried toward the upper pole. In our small series of cases, as stated previously, the majority of parathyroid adenomata have been discovered near the right and left inferior arteries of the thyroid gland. In the

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FIG. 7.—Diagrammatic sketch demonstrating embryologic pathway of superior parathyroid gland which rises from the fourth branchial cleft; anterior and lateral views of the regions in which adenomata of the superior parathyroid glands may be found. Note postero-lateral as well as anterior position of parathyroid tissue, either normal or abnormal, in relation to the thyroid gland, as well as possible incorporation of parathyroid tissue lateral in the carotid sheath and medial to almost midline.

FIG. 8.—Demonstrates embryologic pathways of adult inferior parathyroid gland rising from branchial cleft 3, showing anterior and lateral views. Inferior parathyroid gland or adenoma rising from it may be discovered at any point along this pathway frim 1 cm. or more above sueprior pole of thyroid, down as far in anterior mediastinum as the descent of thymus and lateral from the carotid sheath to midline.

FIG. 9.—(A) Demonstrates possible pathway of mechanically dislocated adenoma rising in superior parathyroid gland, from 1 to 2 cm. above the superior pole of the thyroid gland drawn into the superior posterior mediastinum. (B) Demonstrates path of dislocation of adenoma of inferior parathyroid gland which may be aspirated into posterior superior mediastinum or anterior superior mediastinum, depending on original position of parathyroid gland before enlargement due to development of an adenoma.

absence of adenomata an attempt is made to demonstrate normal parathyroid glands without destroying their circulation, removing them, injuring them, or removing a specimen for biopsy. The primary operation offers by far the best chance of recognition of normal parathyroid glands and even adenomata, for the obvious scarring and loss of normal tissue cleavage will obscure the operative field and render secondary operations technically more difficult. All normal parathyroid glands should be carefully preserved in cases of hyperparathyroidism, for nothing is to be gained by their removal. They are not involved in the production of the clinical syndrome of hyperparathyroidism and chronic tetany will follow if all parathyroid tissue has been sacrificed. Methodical search is made in the region of the right upper pole and postero-lateral surface of the thyroid gland, the region of the pharynx and larynx, the carotid sheath, the retro-esophageal regions between the esophagus and prevertebral fascia, the region between the trachea and esophagus, and the posterior superior mediastinum. If, after this careful dissection, the offending tumor has not been revealed, the same procedure is repeated on the left side.

Because of the fact that at least 80 per cent of the adenomata are found in the cervical region and the posterior superior mediastinum, this dissection is carried out most carefully under direct vision. The posterior superior mediastinum can also be dissected under direct vision through the cervical approach. Nodules in the thyroid gland should be investigated at the time of the cervical exploration after the systematic dissection above described has been carried out, for in two cases in this series the parathyroid adenomata were intrathyroid. Thymic cervical rests should also be investigated, but if no adenomata are revealed in a thymic rest, it should be left in situ; if it is resected, normal parathyroid glands may be removed in the process.

Since parathyroid adenomata vary in size the operating surgeon must be well acquainted with the possible variation in the size and shape of the abnormal parathyroid tissue for which he is searching. The adenomata may vary from .4 Gm. to as much as 120 Gm., and it is only natural that the smaller ones should be more difficult to find than the larger ones. As stated, the largest one in this series weighed 58 Gm. It is also interesting that, as a rule, the adenomata were spheroidal in shape; all of those in our series were spheroidal and averaged around 7 Gm. in weight, being about 1 by 2 by 3 cm. in size. The color in each was likewise a dark blue when seen through the overlying areolar tissue, and when bisected, the cut surfaces were golden brown in color. The presence of degenerated cysts containing old blood was not infrequent, particularly in the larger tumors.

In the instances in which it was necessary to explore the anterior mediastinum it was felt that to do so under direct vision was far superior to blind blunt dissection through the cervical incision. This procedure does not prolong the operation to any great extent and I think should be carried out at the primary operation rather than at a secondary one. There are really no technical difficulties in splitting the sternum down to the third rib, and lateral division of the bone to allow retraction offers no surgical hazard. In one patient in the seventh decade this procedure seemed to be no additional burden of any con-

sequence to the surgical dissection. Finger dissection through the cervical incision is dangerous from the standpoint of tearing not only the blood vessels but also the pleura, and in one instance cited by Cope[29] a pneumothorax developed which was unrecognized, causing the death of the patient. Furthermore, as the adenomata of the parathyroid glands are soft and of varying sizes, palpation is inaccurate. If careful dissection of the neck and superior posterior mediastinum proves fruitless, the sternum should be split and the anterior superior mediastinum dissected under direct vision.

It would seem very important, regardless of whether or not an adenoma of the parathyroid tissue is discovered in the first few minutes of operation, for instance at the right lower pole, to explore all other poles, for in one case in this series a second adenoma, in addition to the one removed from the region of the right lower pole, was found posterior to the left upper pole. They were of equal size and weight. It is, of course, obvious that had this second adenoma been left, the condition of hyperparathyroidism would have persisted. It is our feeling that this subsequent exploration should not include division of the sternum and mediastinal exploration unless there is persistence of the clinical condition. But certainly all the likely locations in the cervical region should be explored at the primary operation.

There has been only one case in this series in which there was a definite malignant change in the parathyroid gland. This condition is very rare. In the literature there are a number of instances in which benign adenomata of the parathyroid glands have been mistakenly diagnosed as malignant because normal parathyroid tissue has seemed to invade the thin capsule of the glands.

The physiologic status and the complications of the individual suffering from hyperparathyroidism must be recognized and understood by the operating surgeon, for in the event of the removal of an adenoma, if the bony changes are predominant and there is high blood phosphatase it is probably the safer procedure to leave a portion of the adenoma transplanted either in the thyroid gland or in the belly of the sternomastoid muscle, for the calcium-hungry bones will drain the calcium ions from the blood stream and tissues to such an extent as to produce critical postoperative tetany. We encountered this in the one case of hypertrophy and hyperplasia of the parathyroid associated with hyperparathyroidism. This condition, which corresponds probably to diffuse hypertrophy and hyperplasia of the thyroid gland, and is comparatively little understood, results from involvement of all the parathyroid tissue present, usually four glands, which are enlarged depending on the degree of hypertrophy and hyperplasia, and histologically show the typical Wasserheller or clear cells. As a rule, as shown in these cases, the gross appearance of such parathyroid glands as have undergone hypertrophy and hyperplasia is not spheroidal, and frequently they have pseudopodial extensions from various portions of the enlarged gland. If this pathologic condition is encountered, partial resection of the large parathyroid glands should be performed, leaving, as pointed out by Albright[12] and others, between 30 and 200 mg. of hyperplastic tissue. Care should be taken to guard the blood supply of the remnant left behind. In the instances in which the renal complications are present with

a normal blood phosphatase, the offending parathyroid tissue such as adenoma may be removed in its entirety.

We have been able to carry out a dissection of sufficient length to enable us to make a complete cervical search and, if this was fruitless, an anterior mediastinal dissection, with complete satisfaction. The anesthetic used is sodium pentothal and curare, this, of course, being supplemented with oxygen administered by means of an intratracheal tube.

PATHOLOGIC ASPECTS

It is beyond the scope of this report to enter into a detailed discussion of the pathologic aspects of tumors and diffuse hypertrophy and hyperplasia of the parathyroid glands. Comprehensive studies by Castleman and Mallory,[24] Norris,[20] and others have been published.

The parathyroid glands, like the pituitary gland, contain many types of cells. The physiologic activity of the different groups, as well as the different types of cells, is at the present time unknown. None of the adenomata of this series consisted of only one type of cell. There was in each an admixture, but in those in which the renal complications alone occurred the chief cells and pale and dark oxyphil cells were in a minority, with the clear, or Wasserheller, cells in a pronounced majority. The reverse was true in those patients having mainly skeletal complications. In these it was our impression that the chief cells predominated and that clear cells, together with pale and dark oxyphil cells, although present, were definitely overshadowed by the preponderance of chief cells. In the one case of diffuse hypertrophy and hyperplasia, the histologic picture showed the gland to be composed entirely of the water clear or Wasserheller cells.

Castleman and Mallory[24] in 1935, in a study of 25 cases representing the patients treated at the Massachusetts General Hospital at that time, evidently had the same thought running through their minds. Their series was divided into hyperplastic and adenomatous groups, and they pointed out that 13 of the latter showed bone lesions, whereas the five clear cell hyperplastic cases fell into the group of those having renal stones without bony changes. Although both their series of 25 cases and ours of 27 are certainly too small to allow one to draw conclusions as to the relation of the cell characteristic of the adenomata removed to the clinical complications, nevertheless, it would seem that such a constant association between Wasserheller or clear cells and renal complications would be more than a coincidence. This question should be held in mind for future investigation. Possibly in a larger series a definite relationship between the two may be established.

Our records are insufficient to date the onset of symptoms accurately, but roughly correspond to those of Castleman and Mallory. It would seem that formation of stones comes first, with bony lesions following only after a period of years. The average duration of symptoms in our patients having only renal stones was much shorter than in the patients with classical skeletal lesions. Castleman and Mallory calculated the duration of symptoms in their patients having renal stones to be 3.2 years as compared to 8.6 years for those with classical bony lesions.

It must be remembered that in reports of earlier cases in the literature the possibility of renal complications was completely overlooked, and the bony lesions of other conditions were often confused with those of hyperparathyroidism. Because of this fact and the inadequate data accompanying earlier reports as to the clinical manifestations, an attempt to correlate the histopathologic changes in the adenomata with the clinical manifestations might well be inaccurate and unreliable. Undoubtedly, in any instance of hyperparathyroidism, regardless of the cellular characteristic of the adenomata, if the condition persists over a sufficient number of years every complication in the clinical syndrome will have developed, whether or not the over-production of parathormone occurred, as a result of proliferation of the chief cells or of the Wasserheller cells. For this reason, also, it may be said that the long-standing cases encountered by the earlier observers, and reported with incomplete clinical data, might not be of value in differentiating between the various cellular activities.

Reports by Churchill,[11] Castleman and Mallory,[24] Rogers and Keating[30] and others on hyperplasia of the parathyroid glands have described pathologic changes observed in association with the clinical manifestations of hyperparathyroidism which are indistinguishable from the clinical syndrome resulting from adenoma of the parathyroid glands. However, it has been pointed out by Castleman and Mallory[24] that the cellular structure of the parathyroid glands in diffuse hypertrophy and hyperplasia is transformed from that observed in the normal parathyroid gland to one composed entirely of the water clear or Wasserheller cells. The analogy between diffuse hypertrophy and hyperplasia of the parathyroid glands and hypertrophy and hyperplasia of the thyroid gland in exophthalmic goiter has been made. Both have been thought to be dependent on some external chemical hormone or nervous stimulation. It is for this reason that it might be interesting to include the report of a patient with exophthalmic goiter and diffuse hypertrophy and hyperplasia of the parathyroid gland.

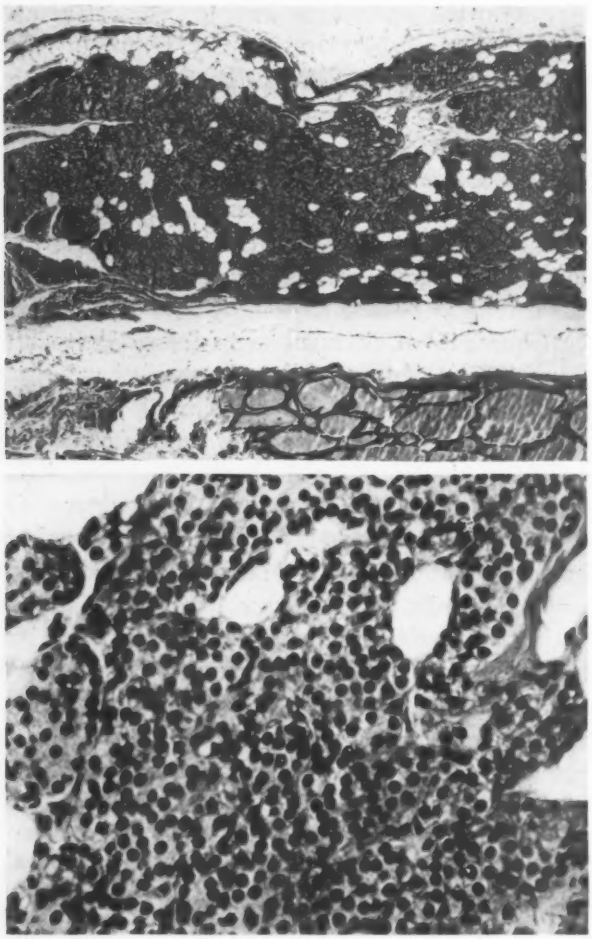
Case Report.—Mrs. S., referred by Dr. Henry M. Thomas, had been treated for hyperthyroidism for 5 years prior to operation at the Johns Hopkins Hospital in 1949. She had been treated with thiourea, thiouracil and propyl thiouracil. She was the wife of an army officer who had been transferred to different posts in this country as well as in Germany. The left lobe of the thyroid was operated on the first time in San Antonio, Texas, and the second time in Virginia. The right lobe had never been dissected. In spite of her two incomplete operations and her constant treatment with thiourea drugs the patient constantly lost weight. The thyroid had increased in size, particularly the right lobe, and there was progressive exophthalmos which had made itself evident during the previous 6 months.

The patient was operated on by me in the Johns Hopkins Hospital on November 21, 1949, at which time partial resection of the recurrence of the left lobe and partial resection of the right lobe were performed. A nodule on the posterolateral surface of the upper portion of the right lobe of the thyroid was observed and assumed to be thyroid tissue. In the gross appearance I was unable to distinguish this nodule from the adjoining thyroid parenchyma. The patient had an uneventful convalescence and has remained well.

When the sections of the thyroid and of the nodule were examined in the Pathological

Department of the Johns Hopkins Hospital, it was found that the nodule consisted of a very much enlarged parathyroid gland which was composed entirely of Wasserheller cells and presented, not only in the gross but microscopically, the appearance of diffuse hypertrophy and hyperplasia of the parathyroid gland.

A



B

FIG. 10.—(A) Low power (x 40) photomicrograph, showing margin of hyperplastic thyroid parenchyma at top and marked hyperplasia of parathyroid tissue at bottom. (B) High power (x 330) photomicrograph, with hyperplastic parathyroid tissue, showing transition from chief to water clear cells.

The patient had no postoperative tetany, probably for the reason that the other parathyroid glands which remained undamaged had undergone some hypertrophy and hyperplasia. Unfortunately, blood studies were not made before operation in the Johns Hopkins Hospital, and I have been unsuccessful in obtaining such data from the hospitals in which she was previously operated upon. A histologic picture of the thyroid and parathyroid in this case is shown in Figure 10. There was, it seemed to us, diffuse

decalcification of the bones of the upper extremities and thorax in this patient as determined by roentgen ray examination.

The literature was searched for any other evidence, experimental or clinical, to support the observation of the effect of the antithyroid drugs on the parathyroid glands. In the British Journal of Experimental Medicine (volume 30) February, 1949, was found a report of hyperplasia of the parathyroids associated with osteitis fibrosa in rats treated with thiouracil and related compounds. This report was made by Malcolm, Griesbach, Bielschowsky and Hall[31] from the Medical School of Otago University, Dunedin, New Zealand. They showed that albino rats treated with thiourea, thiouracil and methylthiouracil for long periods (6 to 18 months) developed pronounced hypertrophy and hyperplasia of the parathyroid glands and, in addition, osteitis fibrosa cystica generalisata (Fig. 11). The parathyroids of these animals were frequently so large that their increased size was apparent to inspection by the naked eye; the parathyroid gland rose above the surface of the thyroid with lobelike projections extending into the neighboring tissue, while the thyroid tissue surrounding the parathyroid showed compression. The microscopic appearance of these glands shows the predominating cell type as a transition from the normal chief cells to the pale oxyphil type and then to the Wasserheller or clear cells. These observations are interesting from two different standpoints; first, the latent effect of the goitrogenic antithyroid drugs on the parathyroid glands, possibly producing hypertrophy and hyperplasia of the parathyroids, and the possible late complications of a serious nature; and second, as far as I am aware, this is the first time that diffuse hypertrophy and hyperplasia of the parathyroid glands has been produced experimentally.

CONCLUSIONS

The 27 cases reported in this series, are few, but the series is not relatively small when one considers that the total number of hyperparathyroid cases diagnosed, to my knowledge, is only 597, which includes those that are published in the entire literature and those learned of through personal communication. The *total number* is indeed small, and for this reason it is felt that the more material recorded in the literature concerning the clinical, pathologic and follow-up data, the greater the opportunities for future observations that may aid in the early recognition and successful treatment of this catastrophic condition. Undoubtedly there are many cases of hyperparathyroidism existing today that remain undiagnosed, and the patients are slowly but surely developing complications which will prove to be irreversible and ultimately fatal.

In practically all of the patients in our series irreversible and irremedial complications have been present and have persisted following operation in which the hyperparathyroidism was corrected. There have been altogether ten deaths in the 26 cases of hyperparathyroidism—excluding the case with carcinoma of the parathyroid gland—with an immediate mortality in one case, or 3.8 per cent (which possibly could be avoided today), and an ultimate mortality of 34.7 per cent. This latter percentage will probably increase in the not far distant future.

Emphasis is laid upon the need for early diagnosis before renal and

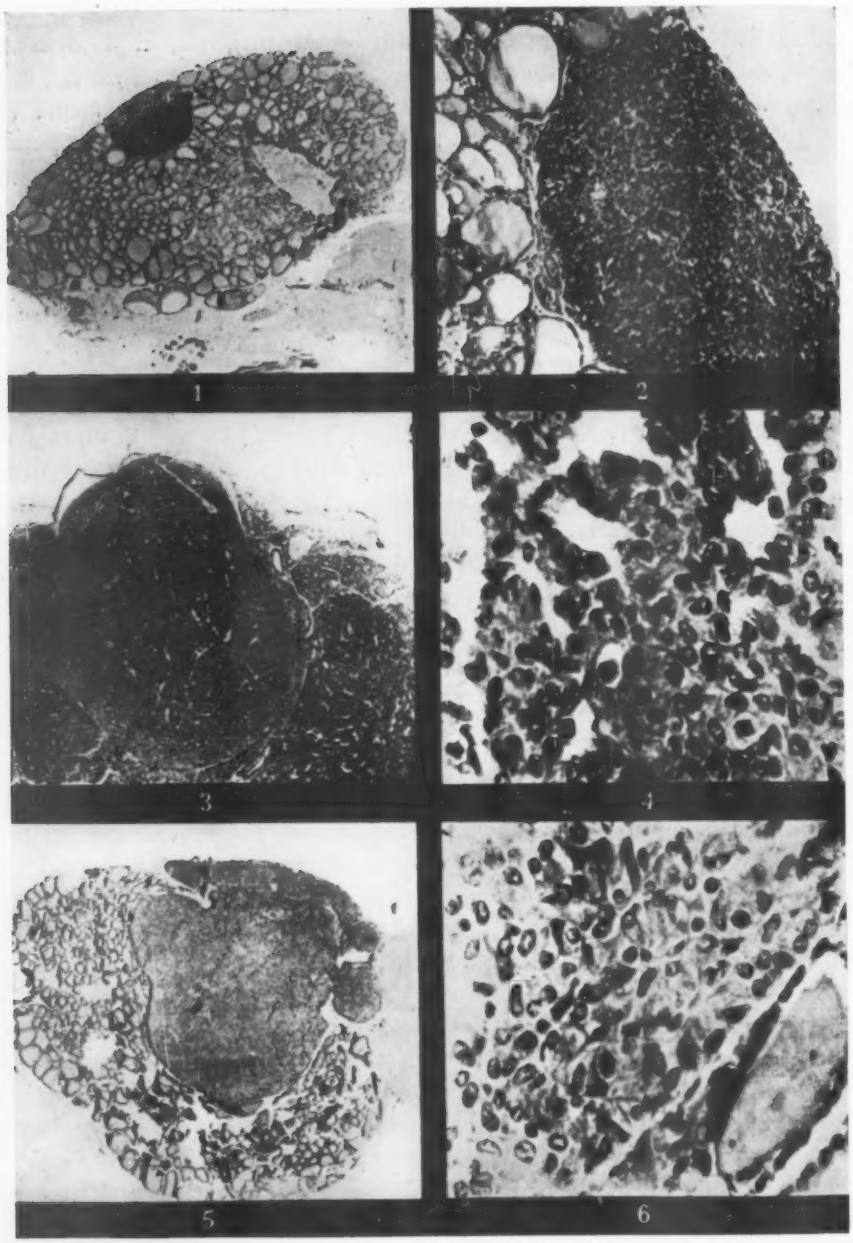


FIG. 11.—Photomicrograph, reprinted from British Journal of Experimental Medicine, showing (1) normal parathyroid and thyroid glands in albino rats; high power of normal (2). In 3 is shown the bulging hyperplastic parathyroid gland beyond the limitation of the thyroid gland; In 4 is shown high power of microscopic picture, demonstrating transition from chief to water clear cells and after 18 months of administration of antithyroid. In 5 is shown an enormous hypertrophy and hyperplasia of parathyroid gland, microscopic picture of which is shown in 6.

osseous complications develop in view of the postoperative course of individuals afflicted with the complications of hyperparathyroidism. The necessity for closer study of the normal parathyroid gland in its usual position is stressed and attention is again called to the embryologic pathways and mechanical displacements which may exist. As pointed out, the recognition of the character of these adenomata in contradistinction to hypertrophy and hyperplasia constitutes one of the fundamental conditions in the operative correction of this syndrome. It is necessary for the operating surgeon to familiarize himself not only with the various locations in which normal and abnormal tissue may be found, but also to be cognizant of the physiologic effects of the excessive secretion of parathormone and the possibility of the development of postoperative tetany, depending upon the character of the complications.

Although it is attempting conclusion that cannot be drawn because of insufficient reported studies, the possible correlation of the complications associated with the particular cell type which predominates in the various adenomata, and instances of hypertrophy and hyperplasia, is discussed.

Attention is called to the possible effect of the antithyroid goitrogenic drugs, administered over a long period of time, on the parathyroid glands.

Determination of the calcium in the cerebral and spinal fluid, it is hoped, will prove to be of diagnostic importance to differentiate the hypercalcemia of hyperparathyroidism from other conditions in which hypercalcemia is present.

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DISCUSSION—DR. H. P. ROYSTER, Philadelphia: We have 19 patients with pathologically proved hyperparathyroid disease. We have found many problems in this particular disease and have been interested in it since the first patient was operated upon in 1933. I believe our chief problem at the present time is in making the diagnosis in a borderline case; the borderline case is usually presented to us from the urologic clinic, where blood calcium, phosphorus and phosphatase examinations have been done, not only once but perhaps several times, on a patient who comes in with a renal stone. We have found, as Doctor Rienhoff suggested, and as earlier described by Doctor Cope, that the best method is to take repeated examinations of the blood and not be satisfied with the diagnosis or the lack of it, on a single examination. We have also done renal calcium examinations on most of these patients when we have been somewhat in doubt as to whether or not they had the disease. We have learned, of course, not to depend entirely on that, for we have found a number of patients who exhibited increased excretion of calcium in the urine when every other finding, except perhaps one or two blood chemistry examinations, was normal. We have also found out that many patients who have diseased kidneys may excrete more calcium than the normal; one would expect the opposite to be the case. We have two patients who have had nephrostomy with a tube in one kidney collecting all the urine, and with bladder urine being excreted to account for the excretion of the other kidney, and it was noted that the more diseased kidney would excrete a higher percentage of calcium than the less diseased organ. This, we think, has made it possible for us to make errors in diagnosis, and a number of patients have been operated upon erroneously because of that finding. Consequently, we no longer put as much faith in the high renal excretion of calcium. We are cognizant of the importance of renal complications because, after all, that is the cause of death in these patients. So far, of the 19 patients we have had, only one has died, although several others have badly damaged kidneys. One patient, 55 years old, remained in tetany over a five-year period after an adenoma was removed in 1934, and she is still on the verge of tetany. Her renal function over a period of 15 years has remained stationary, and she pursues a normal life working as a visiting nurse. The low blood calcium of the tetanic state may have been beneficial to the kidneys.

We also think that many individuals who have excretion of calcium above normal or on the borderline do much better, and they say they feel better if calcium is eliminated from the diet. There has been a great deal of discussion of this, but certainly if there is an increased amount of calcium going through the kidneys it is wise not to add any more than would be necessary in a normal diet.

DR. FRANK H. LAHEY, Boston: Doctor Rienhoff asked me if I would discuss this paper, and I said I would if I could add something to it. I am sure that what I attempt to add will probably be in the body of his paper which he has not had time to read.

We have not had a large experience with this condition. I too want to add a word of appreciation to the group at Massachusetts General Hospital, notably concerning the earlier stages of their work; to Dr. Joseph Aub who, incidentally, was one of the first to call attention to the fact that there is an intense decalcification in patients with hyperthyroidism; to Doctor Albright, to whom we all owe a great deal for the fundamental work which has been done on the normal and the abnormal physiology of the parathyroids, and to Doctor Cope and Doctor Churchill for their significant contribution to the surgical management of this problem.

I would like to emphasize again what we have stressed repeatedly, that is, the diagnosis is too often made too late. This disease is not characterized by very obvious symptoms, and when they are obvious, the undesirable and irreversible changes have taken place. Not an unimportant one is collapse of the vertebral body. It is too late to do any-

thing in the way of changes when decalcification has existed for such a time that vertebral bodies have collapsed and established nerve root pressure and round backs and, of course, when calcification has taken place in the kidney, as has been shown by Doctor Rienhoff, that is an irreparable and irreversible state. It is unfortunate that we do not have a simple quantitative test for hypercalcemia. We do have a simple test, the Sulkowitch test; this is not of much dependable value in the hypercalcemias but is applicable in the hypocalcemias. With patients who have hypoparathyroidism this test is valuable in demonstrating the maintenance of proper levels of calcium. Unfortunately, it is of little value in hyperparathyroidism, because everyone is on such a high calcium diet. If the internists in particular have in mind in their studies of general medical states that weakness, kidney stones, arthritic symptoms, nerve root pressure, and, when roentgenograms are available, decalcification, should prompt one to do chemical studies which will indicate the presence or absence of this disease, then we will make the diagnosis earlier and will obtain, I think, much better end results. One should be suspicious of hyperparathyroidism when calcium levels are 10 or higher, when phosphorus levels are 3 or lower, and when phosphatase levels are 8 Bodansky units or higher. I am sure Doctor Rienhoff has called attention in his paper to the necessity of repetition of these studies to establish their accuracy.

There are two or three clinical points I would like to make, because everyone who has dealt with these has had problems with them. I noticed in a recent issue of the Mayo Clinic Proceedings the report of two intrathyroid parathyroid adenomas. We have had a similar experience in finding intrathyroid parathyroids and, as one searches for possible hyperfunctioning adenomas, one must have this in mind.

Doctor Cope called our attention to a very valuable clinical measure, that is, if you cannot find the hyperfunctioning adenoma, you must demonstrate, if possible, all the parathyroids and then, if one is missing—although they do vary in number—search that area particularly. I can remember very well searching for one of these; I had turned my back to discuss the different places where parathyroids were found, and Dr. Ralph Adams exposed the inferior thyroid artery and demonstrated a branch leading down into the superior mediastinum, which led us to a hyperfunctioning parathyroid adenoma just beneath the sternum.

Finally, I would like to call attention to a point which we have repeatedly stated, which plays an important part in the postoperative comfort of these patients, that is, that the majority of these patients will have postoperative tetany, and it is a terrific ordeal to go through if we wait until they get into the acute phases of postoperative tetany. It is frightening to the patient and certainly frightening to his family. We must be prepared to meet postoperative tetany by the various methods which are now available, before it occurs. We have followed patients who have drops in calcium from 18 to low levels within 24 hours after removal. We must remember that, as in many other similar situations, the production of parathormone is largely in the hyperfunctioning adenoma, and if you look at the other parathyroids they are pale, fat-infiltrated and atrophic, and in many cases it will take up to three months to regain ability to produce enough parathormone to maintain the patient in parathormone equilibrium.

This is an interesting subject and, like many of the others, we need to be reminded repeatedly of the possible presence of these diseases, and any group that is interested in them will find them.

Dr. William F. Rienhoff, Jr., Baltimore (closing): I wish to thank Dr. Frank Lahey and Doctor Royster very much for their interesting discussions. Because I wished to stay within my time limit there were a member of points brought out in the body of the paper which I was unable to mention even briefly, but one additional fact I wish to point out is that in only one patient in this series was there a coincidental duodenal ulcer which gave rise to bleeding before the parathyroid adenoma was removed. There was no bleeding afterward and apparently the ulcer healed. The association of duodenal ulcer with hyperparathyroidism has been mentioned by others in the past, particularly by Rogers and Keating, in a series of cases reported from the Mayo Clinic.

POTASSIUM DEFICIENCY IN SURGICAL PATIENTS: ITS RECOGNITION AND MANAGEMENT*

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THE CLASSICAL WORK of Gamble[1] laid the foundation for the adequate treatment of sodium deficiency, and we are indebted to Coller and Maddock[2] for placing this knowledge on a practical basis for the care of surgical patients. Although sodium deficiency is extremely important, Darrow's[3] recognition and management of potassium deficiencies in pediatric practice deserves equal consideration by the practical surgeon.

Our interest in potassium deficiency in surgical patients stems from observations made on dogs suffering from dehydration, chloride deficit and alkalosis.[4] In these animals, infusions of 0.9% sodium chloride solutions restored sodium and chloride plasma concentrations to normal, with correction of alkalosis, but were accompanied by a lowering of plasma potassium levels. For correction of mixed water and salt depletion, we therefore advised balanced infusion solutions of NaCl + KCl + $CaCl_2$ + $MgCl_2$ instead of simple saline (Van Slyke and Evans).[5] In 1947, we were asked to advise treatment of a patient with intractable alkalosis not corrected by adequate saline therapy, caused presumably by excessive adrenal cortical hormone therapy resulting in a low plasma potassium concentration. These observations were followed by intensive investigation in the dog; potassium depletion was produced experimentally by a low potassium and high sodium chloride intake, plus glucose diuresis plus daily injections of 10 mg. of desoxycorticoacetate. Severe alkalosis then followed intra-peritoneal infusions of sodium bicarbonate solutions. When saline was administered to such animals, despite dangerously low plasma and intracellular potassium, alkalosis was readily corrected. These experiments did not fit in with our clinical observations on the above-mentioned patient and will be reported in detail later with my colleague, Dr. K. K. Van Slyke.[6]

Since 1947, we have studied electrolyte patterns in many surgical patients where mixed water and salt depletion existed, and have made observations on potassium deficiency and its correction in these patients. The present report is concerned with a description of our experience with certain of these patients who exhibited plasma potassium deficiency, they being selected for presentation because collectively they represent the type of surgical patient in whom potassium deficiency is most likely to be encountered by the average surgeon.

† With the technical assistance of Carolyn Martin and Rachel Fitzpatrick, Surgical Research Laboratories, Department of Surgery, Medical College of Virginia, Richmond, Virginia.

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In general, remarks are confined to a brief description of the cause of potassium deficiency in surgical patients, its recognition, and some practical details of the management of this deficiency.

With the recent publication of the carefully collected and annotated review of all pertinent literature on potassium deficiency in surgical patients by Randall, Habif, Lockwood and Werner,[7] it is unnecessary in this paper to do more than refer to certain published works in discussing this subject.

THE CAUSE OF POTASSIUM DEFICIENCY IN THE SURGICAL PATIENT

Sodium and potassium are the chief cations of the human body, sodium residing chiefly in the plasma and extracellular fluids, whereas normally almost all the potassium (98 per cent) is in the intracellular fluid, in muscle and liver

TABLE I.—*G. I. Tract Losses M. E. Q. Per Liter.*

	Na	K	Cl
Gastric	60.4	9.2	84.0
	9-116	0.5-32.5	7.8-154.5
Small Bowel	111.3	4.6	104.2
	82-147.9	2.3-8.0	43-137
Ileostomy	129.4	11.2	116.2
(Recent)	105.4-143.7	5.9-29.3	90-136.4
Ileostomy	46	3.0	21.4
(Adapted)			
Cecostomy	52.5	7.9	21.4
Bile	148.9	4.98	100.6
	131-164	2.6-12	89-117.6
Pancreas	141.1	4.6	76.6
	113-153	2.6-7.4	54.1-95.2

Average figures and ranges observed for sodium, potassium and chloride ions from biliary and pancreatic fistulas, together with ranges observed in urine and transudates (from Lockwood and Randall, Bull. New York Acad. Med., p. 234, April 1949.)

cells. Formerly, it was believed that the cell membrane was practically impermeable to potassium and sodium with very little, if any, transfer of these cations from one space to the other. It is becoming more widely recognized that under certain conditions of dehydration and salt deprivation or loss, transfer of some of the cell potassium to the extracellular space may occur, to be replaced under certain conditions by the ingress of sodium from the plasma or interstitial fluids to the cell.

It has been recognized for some time that inordinate loss of body fluids by prolonged vomiting or prolonged diarrhea leads to considerable wastage of sodium and chloride. It must be emphasized here, however, that vomitus and drainage fluids, and those from the biliary and upper intestinal tract, contain large amounts of potassium as well as sodium (Table I). When sodium and potassium is lost in this manner, the normal kidney tends to conserve sodium by almost complete tubular reabsorption of sodium (Van Slyke and Evans[5]); the excreted urine contains almost no sodium. Unfortunately, no such renal mechanism appears to act to conserve body potassium, so even in states of severe potassium depletion this cation continues to be lost in the urine. Indeed, after accidental or surgical trauma, sympatho-adrenal discharge

(Selye's "alarm reaction") may result in extraordinary loss of potassium by the urinary route, whereas because of the effect of the adrenal cortex hormone on the renal tubule, sodium is retained in an abnormal fashion.

If gastric and small bowel fluids (along with those from the liver and pancreas), as well as urine, contain so much potassium (as well as sodium and chloride), it follows that if a patient loses large amounts of these fluids by excessive or prolonged vomiting, gastric or intestinal suction, or diarrhea or alimentary tract fistulas, and potassium is not replaced in adequate amounts by oral or intravenous route, potassium deficiency to some degree must ensue. Individual case reports are given below to indicate the importance of the potassium deficiency states met with commonly by the clinical surgeon (inanition due to esophageal carcinoma, pyloric obstruction (ulcer), fecal fistula, protracted diarrhea, intestinal obstruction, paralytic ileus, severe trauma followed by gastric suction; to which is added discussion of the effects of adrenal cortical hormone used by some surgeons to counteract traumatic shock).

THE RECOGNITION OF POTASSIUM DEFICIENCY IN THE SURGICAL PATIENT

Generally speaking, the signs attributed to potassium deficiency in the patient refer to severe grades of this deficiency. These signs, (1) extreme muscular weakness, (2) paralysis of accessory respiratory muscles, (3) aphonia, and (4) coma, in our experience are not common, although they have been seen in some patients. Positive electrocardiographic findings (low amplitude of the T waves, lengthening of the Q-T interval) and a serum potassium level of 2.6 MEQ or below enable the surgeon to make the diagnosis. These, however, are signs and symptoms of *extreme* potassium deficiency. More commonly, the diagnosis of potassium deficiency in the surgical patient depends upon an intelligent evaluation of the clinical state likely to be associated with this deficiency, that is, dehydration and salt loss produced by excessive vomiting, diarrhea, or other causes (see above). In our experience, if an alkalosis (CO_2 content above 60 volumes per 100) persists in such patients after adequate hydration and sodium chloride therapy, potassium deficiency is to be strongly suspected. The diagnosis *may be* confirmed by analysis of the plasma or serum for potassium level. A serum potassium level below 3.5 MEQ/liter is confirmatory, but unfortunately *potassium deficiency can exist in the presence of normal plasma potassium levels* (see case C.M. below) and secondly, equipment is not generally available to the average surgeon for these analyses.

For practical reasons, therefore, the surgeon is advised strongly to pay close attention to *uncorrected alkalosis after adequate water and sodium chloride therapy* and to suspect potassium deficiency if this abnormality exists.

POTASSIUM DEFICIENCY IN SURGICAL CONDITIONS

Methods. All blood samples, except in an emergency, were drawn in the early morning before fluids were started or before food was taken. All blood samples were heparinized and, for bicarbonate determinations, drawn and kept under oil. Sodium and potassium determinations were done with the

Perkin-Elmer flame photometer, which, in our hands, has a reproducible accuracy of 1.5 to 3.0 per cent. Chloride ion determinations were made by the method of Van Slyke and Sendroy.[8] Bicarbonate concentration as CO_2 content was determined by the method of Van Slyke.[9] In the charts presented in this paper, these determinations were made daily.

Normal values in this laboratory are: sodium 140 to 145 MEQ, potassium 3.8 to 4.5 MEQ, chloride 101 to 107 MEQ and CO_2 content, 25 to 28 MEQ, all per liter.

I. Continuous Gastric Drainage.

Surgical patients recovering from operation (appendectomy 5, gastrectomy 1, perforated ulcer 3, stab wound of chest 1) with an indwelling gastric tube for constant Wangensteen suction were studied to learn how long continuous gastric suction must be applied before potassium deficiency is exhibited by a fall of plasma potassium below 3.5 MEQ/L. In general, these observations, coupled with those made in an analysis of data from other patients suffering from potassium deficiency associated with loss of gastric fluids, lead us to believe that continuous gastric drainage, without adequate potassium replacement, *after four to five days* may result in potassium deficiency. These observations confirm those reported by Dr. Lockwood's group—if gastric suction must be continued beyond *four to five days,* provision should be made for the intravenous infusion of adequate amounts of potassium salts.

The following case reports are given in some detail to illustrate the occurrence of potassium deficiency in certain common surgical conditions.

II. Inanition Due to Esophageal Carcinoma (Case Report).

E.W. (B75042). This 47-year-old woman entered the hospital with a diagnosis of esophageal stricture caused by a lye burn at 4 years of age. Swallowing of food was possible until 5 months before entry. At that time, dilatation of the esophagus was done, and this was repeated one month later. At this second dilatation, rupture of the esophagus occurred, followed shortly after by a right pleural empyema which was drained surgically. For one month prior to the last hospital entry there had been considerable difficulty in swallowing solid foods, with complete obstruction of the esophagus one week before she was admitted. Esophagoscopy and biopsy of the stricture zone in the upper third of the esophagus revealed a carcinoma, grade II. On November 8, 1949, Dr. I. A. Bigger performed an esophageal resection, anastomosing the upper esophagus with the stomach. The only abnormal blood chemistry finding preoperatively was a CO_2 content of 32 MEQ. The postoperative course was smooth until the second day, when the patient became very weak and listless. Potassium deficiency was demonstrated by plasma potassium level of 2.7 MEQ (See Fig. 1). Potassium therapy of 1 Gm. KCl in 1 liter saline was given that day, and repeated on the third and fourth postoperative days. As this was not sufficient to correct potassium deficiency 5 Gm. of KCl in 2000 cc. 5 per cent glucose in distilled water was given on the fourth postoperative day. On the following days, 4 Gm. of KCl in 2000 cc. Ringer's solution was given daily, and 3 Gm. KCl given daily through an indwelling jejunostomy tube. By the eleventh postoperative day the plasma potassium level was normal (4.3 MEQ). (See Figure 1.)

Comment. Potassium deficiency does undoubtedly occur in many patients who fail, for one reason or another, to take adequate food by mouth. It should

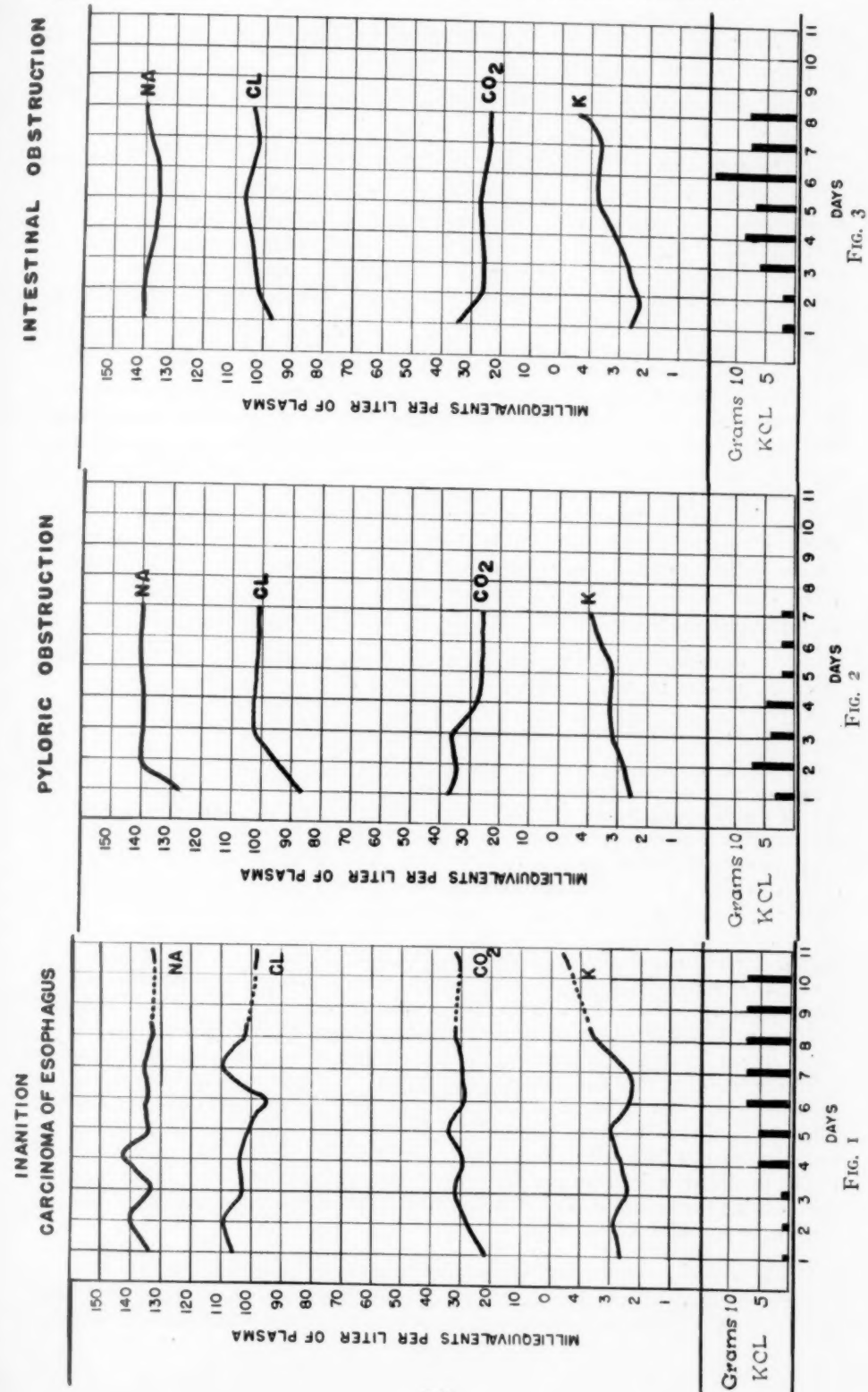

FIG. 1

FIG. 2

FIG. 3

be suspected in all patients with esophageal stricture due to lye burns or carcinoma. It is interesting again to note in this patient the preoperative abnormally high CO_2 content of the plasma (32 MEQ), associated with a relatively normal plasma chloride level (98 MEQ).

III. Pyloric Obstruction (Ulcer), (Case Report).

S.E.M. (B59781). This 68-year-old man was admitted on Dr. I. A. Bigger's service February 13, 1949, with a diagnosis of peptic ulcer. He had been able to take very little food for several weeks, and had lost considerable weight. Preoperative roentgen ray examination demonstrated almost complete obstruction at the pylorus. There was free hydrochloric acid and blood in the gastric washings. Because of a hemoglobin of 11 Gm. he was given one blood transfusion before operation, along with adequate intravenous fluids of Ringer's solution and amino acids. The only abnormal blood chemical findings preoperatively was a high CO_2 content of 32 and 34 MEQ.

He was operated on by Dr. Bigger on February 16, 1949; a subtotal gastrectomy was done. The stomach lesion proved to be benign. Because the gallbladder contained several large stones, these were removed and a cholecystostomy performed. The immediate postoperative period was satisfactory. Fluid and electrolyte balance was maintained largely with adequate amounts of amino acids and 0.9 per cent NaCl solutions and 5 per cent dextrose in water, resulting in a satisfactory urine output. However, on the third postoperative day, the blood pressure fell to shock levels (90/50), the patient was extremely drowsy, difficult to arouse, speaking only in a whisper. Serum chlorides were then 87 MEQ. After 1500 cc. of Ringer's solution, these had risen to 97 MEQ, with a plasma sodium of 129 MEQ, and plasma potassium of 2.7 MEQ. Because of this low plasma potassium level, he was given two infusions of 250 cc. of 0.2 per cent KCl solution three hours apart. After the second injection he began to talk rather freely and appeared much stronger, although his blood pressure still remained at 85/50. Four hours later, he was given the third 250 cc. of .2 per cent KCl in Ringer's solution. His blood pressure rose to 104/65, and later that day to 110/66, after which it never fell again to shock levels. Potassium chloride was injected in the indwelling gastrostomy tube at a dose of 500 mg. every 4 hours, this later being changed to 300 mg. every third hour. On the fifth postoperative day, the patient was considerably improved, the blood pressure being then 140/80 and maintained. His general body strength appeared normal for a patient who had undergone a serious operation. That day he was given 1000 cc. of Ringer's solution to which 2 Gm. of KCl had been added. The patient was allowed to sit at the side of the bed with the gastric tube clamped for varying periods. The gastric tube was removed on the sixth postoperative day but KCl continued, 300 mg. every 3 hours with small amounts of milk. On the seventh postoperative day the patient was allowed small amounts of Sippy II diet, the next day a soft diet, after which time he was given a normal diet. Potassium chloride therapy was continued until the tenth postoperative day, 300 mg. every third hour, after which he no longer needed potassium therapy, being able to take a full diet.

Figure 2 illustrates the chemical findings in this patient from the time potassium deficiency was discovered until potassium therapy was no longer necessary. It will be seen from this chart that simple potassium therapy, with fluid and salt needs corrected by Ringer's solution, quite easily corrected the deficit, this treatment resulting on the tenth postoperative day in normal plasma figures for sodium, chloride, CO_2 and potassium. In this particular patient, the gastric drainage was minimal (averaging only 275 cc. per day) making the task of potassium replacement quite easy, because food could be given early in the postoperative period.

IV. Intestinal Obstruction (Case Report).

F.W. (B60094). This 68-year-old woman entered the hospital on February 26, 1949, with a history of swelling of the abdomen for one week with sharp, intermittent, abdominal

pain for two days; no vomiting and poor bowel movements for one week. Examination showed marked abdominal distention. The patient was quite drowsy and disoriented. A diagnosis of large bowel obstruction was made. She was operated on by Dr. Bigger, who reduced a volvulus of the large bowel; there was moderate distention of the small bowel, but no other lesion.

The preoperative blood examination showed the following plasma levels; sodium 140 MEQ, potassium 2.6 MEQ, CO_2 35 MEQ, chloride 97 MEQ. Moderate potassium deficiency was diagnosed and immediately postoperatively the patient was given 2 Gm. KCl in 1000 cc. of Ringer's solution. This was repeated the following day. On the second postoperative day, plasma potassium was low, 2.4 MEQ, so 6 Gm. KCl were administered in 2 liters of Ringer's solution. Following this the patient became more alert, and moved her arms and legs in a restless fashion. The third postoperative day the same dose of KCl was given, with KCl given orally through the indwelling gastric tube, 300 mg. every third hour.

Figure 3 shows the blood chemistry findings with a record of KCl administration for the first 8 hospital days. During this period, 63 Gm. KCl was required to bring the plasma potassium to a normal level. During this time she received, by the intravenous route, 112 Gm. NaCl. When KCl was given intravenously it was administered as 2 to 5 Gm./liter Ringer's solution. On the eighth postoperative day, although the patient was shaky and weak, she was out of bed and took food by mouth fairly well. She was discharged from the hospital on the fifteenth postoperative day.

V. Fecal Fistulas (Case Report).

L.P. (B58124). This 66-year-old man entered the hospital on May 1, 1948, with a diagnosis of unexplained abdominal pain. He had suffered for years from chronic Buerger's disease, for which bilateral amputation had been done. He had been treated for gastric ulcer with Sippy diet for 3 months prior to entry. Examination revealed tenderness in the epigastrium and around the umbilicus. He was watched conservatively for 24 hours, but because of a rise in white blood cell count and localizing tenderness in the right lower quadrant, an operation was deemed advisable. A preoperative diagnosis of possible perforated peptic ulcer was made. At operation, no lesion was found, but an appendectomy was done. The postoperative course was not unusual. The sutures were removed on the eighth postoperative day when it was noted that the wound was moderately edematous. A few hours after the sutures had been removed, there was complete dehiscence of the wound, so the patient was taken to the operating room for secondary closure. Some small bowel was found protruding into the wound and on attempts to release this, a 1 cm. perforation occurred. This was closed, the bowel returned to the abdominal cavity, and the wound closed. The wound did not heal well and this postoperative course was accompanied by considerable fever. On the sixth postoperative day he began to drain considerable amounts of fecal material from the operative wound. By the next day, copious fecal drainage was taking place and from then on, large amounts of fluid were lost from the fecal fistula.

We began studying this patient seriously from a blood chemical viewpoint 2 days later. Because urine collections were excellent we were able to calculate fluid and salt requirements quite easily. Owing to the large amounts of fecal drainage, this patient required from 2 to 3 liters Ringer's solution per day. He was able to take a fairly normal diet by mouth. Despite the daily intake of .6 to 1 Gm. KCl, it will be seen from Figure 4 that so long as the fecal fistula was draining profusely, we were never able to achieve a satisfactory plasma potassium level. Once the fistula closed the potassium level rose to normal in a few days.

Comment. At no time did this patient illustrate signs of even moderate potassium deficiency. It will be noted from the chart (Figure 4) that despite the low plasma potassium levels, it was not associated with alkalosis. The

sodium and chloride plasma levels were essentially normal throughout the course of the chemical observations on this patient.

We have studied three patients with fecal fistula. All involved the lower ileum. In each patient there was profuse fecal drainage, accompanied by low plasma potassium levels, but normal sodium and chloride levels; at no time was there alkalosis in any of these three patients. In no instance, despite large amounts of potassium by mouth, was a normal plasma potassium level achieved until the fistula was closed surgically. These cases indicate that in fecal ileal fistula, despite low plasma potassium levels, (and presumably low cell potassium) alkalosis does not result.

VI. Paralytic Ileus (Case Report).

C.M. (B73189). This 54-year-old woman entered St. Philip Hospital on September 20, 1949, with a diagnosis of pelvic inflammatory disease. She had complained of lower abdominal pain for 7 days. There had been no nausea, vomiting or diarrhea. There had been a questionable weight loss for the past few years. She had noted progressive weakness for 4 days prior to entry. Examination showed moderate distention of the abdomen, with generalized abdominal tenderness, most marked in the lower quadrants. Abdominal auscultation revealed few peristaltic sounds. Pelvic examination demonstrated a mass in the left vault. A diagnosis of chronic pelvic inflammatory disease with pelvic abscess and intestinal obstruction was made. Gastric suction was carried out for 8 days, fluid and salt balance being maintained with intravenous therapy, with a satisfactory urine volume, and salt output being maintained. The patient was operated upon September 30, 1949, at which operation pelvic inflammatory disease, right tubal abscess and uterine fibroids were found. There was no evidence of intestinal obstruction. There was evidence of moderate paralytic ileus of small bowel; a total hysterectomy and a right salpingo-oophorectomy was performed.

Postoperatively, fluid balance was maintained by at least 1 to 2 liters of Ringer's saline daily, accompanied by adequate urine and sodium output, with Wangensteen suction drainage.

A plasma potassium level of 3.6 MEQ on the day of operation indicated satisfactory potassium balance, but chemical examination of a rectus abdominis muscle biopsy taken on the day of operation and reported on the fifth postoperative day showed a severe body potassium deficiency (muscle potassium of 0.283 Gm./100 Gm. and sodium of 0.09 Gm./100 Gm., wet weight.) On this fifth postoperative day, a second plasma determination showed extreme potassium deficiency with a level of 1.6 MEQ (on the first postoperative day, plasma potassium was normal, 4.3 MEQ). This low plasma potassium was associated with fairly marked abdominal distention; on auscultation, no peristaltic sounds were heard. Potassium therapy was started, 4 Gm. KCl being given in 2 liters of Ringer's solution. In about 12 hours, the abdominal distention was less. The patient was given, thereafter, 2 Gm. KCl in each liter of intravenous Ringer's solution and 5 Gm. KCl each 12 hours by mouth. This was continued until the eighth postoperative day. Then, because the plasma potassium level was low, and the plasma bicarbonate high, the potassium intake by mouth was increased to 30 Gm. a day.

Comment. Despite this large intake of potassium, it will be seen from the chart (Fig. 5) that even as late as the eleventh postoperative day (day 7 on chart), a normal plasma potassium or bicarbonate concentration was not reached. This, we believe, is quite easily explained by the extreme potassium deficiency discovered by chemical analysis of the muscle biopsy.

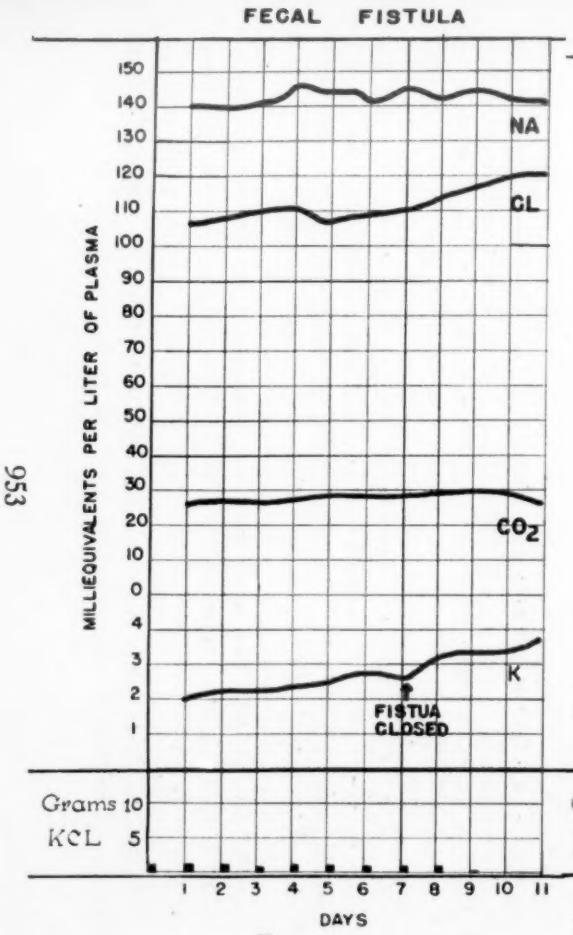

FIG. 4

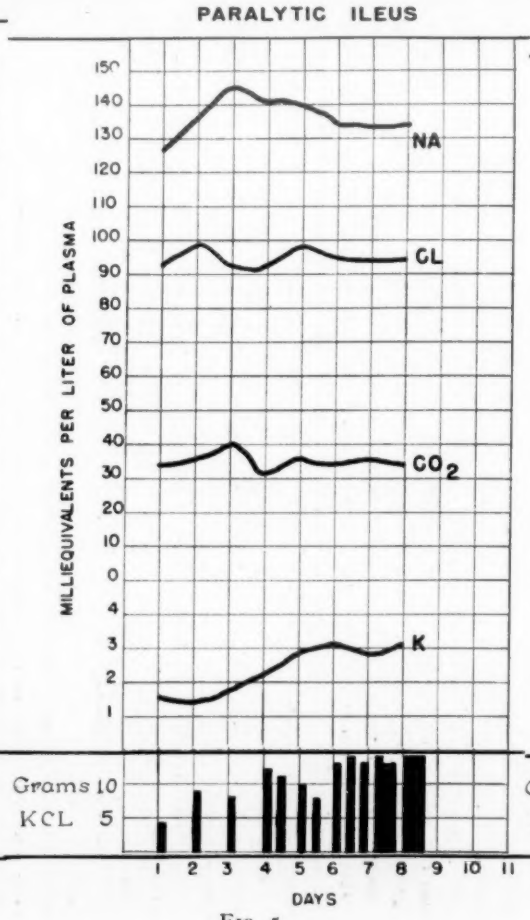

FIG. 5

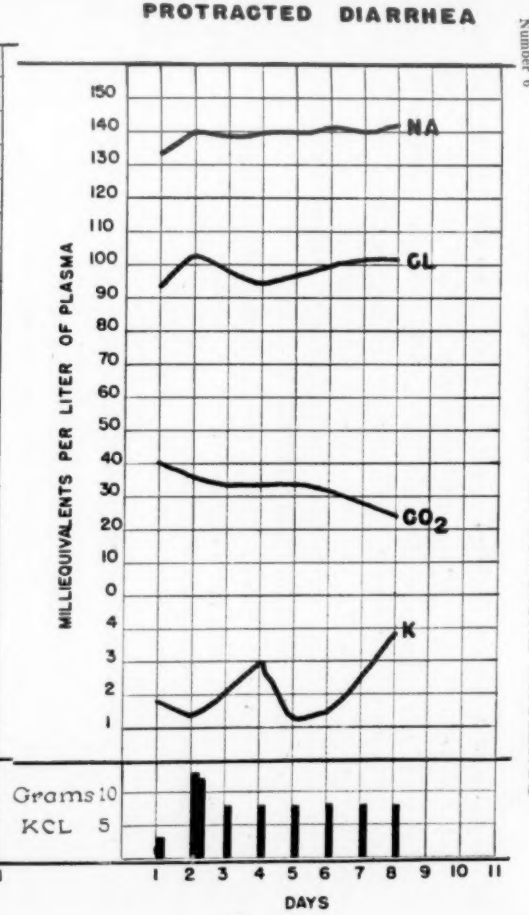

FIG. 6

Data on this patient indicate again that even in severe muscle potassium depletion, normal serum potassium levels may exist. The only preoperative indication of potassium deficiency in this patient was the unexplained high plasma CO_2 level, 35 and 37 MEQ, and in the presence of normal plasma chloride, 97 and 98 MEQ.

VII. Protracted Diarrhea (Case Report).

J.M. (B71141). This 67-year-old man was well until one year prior to hospital entry, when he underwent a resection of the ileum for mesenteric thrombosis at another hospital. At that operation, accompanying the ileal resection, a jejuno-cecal anastomosis was made. Since the operation, the patient had frequent bowel movements of a watery consistency varying from 12 to 25 each day. Shortly before this hospitalization the number of bowel movements had diminished steadily to 2 to 5 per day. Increasingly, he had noted bodily weakness, and had become bedfast. On examination the patient was found to be extremely weak. He was seen by Dr. Richard Neubauer on the medical service, who suspected potassium deficiency, along with general malnutrition. Plasma potassium was 1.7 MEQ. The patient was given 3 Gm. KCl in 500 cc. 5 per cent dextrose solution. The next day, because of extreme muscle weakness, he received 500 cc. 5 per cent KCl (5 Gm./100 cc.) solution. A muscle biopsy (gastrocnemius) was taken, and chemical analysis in our laboratory revealed extreme potassium deficiency (K = 0.162, Na = 0.107 Gm. per 100 Gm. wet weight). Thereafter, the patient was given 8 Gm. KCl for the next 6 days and by the eighth day of study plasma potassium had risen to normal level and alkalosis was corrected (See Fig. 6). Convalescence was prolonged by multiple vitamin deficieneies, hypocalcemia and hypoproteinemia, but by the tenth hospital day, muscle tone and general body strength was greatly improved.

Comment. Studies on this patient indicate that in surgical conditions, such as chronic ulcerative colitis, tuberculous enteritis, intestinal polyposis, etc., when severe diarrhea is protracted, potassium deficiency can become a hazard to the surgeon and patient, and should be corrected before operation is contemplated.

VIII. Severe Trauma; Ruptured Ileum; Shock.

S.P. (B59200). This 19-year-old boy was admitted to the hospital on January 20, 1949, after having been in an automobile accident. On entry, he was in severe shock. He received 1000 cc. of whole blood, which brought the blood pressure to 112/70. Roentgen ray films showed bilateral fracture of the superior and inferior pelvic rami. Twenty-four hours later there was considerable abdominal spasm, with the patient complaining of severe pain in the abdomen. Examination showed the abdomen to be tense, with no intestinal sounds being heard. The blood pressure was normal.

Operation was carried out by Dr. I. A. Bigger, who found the lower portion of the ileum torn loose from its mesentery, gangrenous, and with a small leak. Resection of the terminal 2 feet of ileum and proximal colon up to the middle colic artery was done with a side-to-side ileocolic anastomosis, the patient receiving 2000 cc. of whole blood in the operating room. His postoperative course was extremely stormy, but by the second postoperative day, he began to show improvement, although his urine output was only 600 cc. for the first 24 hours. The patient was given large amounts (up to 5300 cc.) of Ringer's solution daily, these large amounts being required because the gastric suction withdrew from 3 to 4 liters of gastric fluid per day. By the fifth postoperative day, his condition appeared quite good. However, blood chemical determinations on the fifth postoperative day showed evidence of severe alkalosis. On the first postoperative day, blood chemistry

studies showed sodium 152 MEQ, chloride 96 MEQ, CO_2 content 50 MEQ, and potassium 4.7 MEQ. Thereafter, daily determinations of sodium, chloride and CO_2 were made, determination of plasma potassium not being done again until the eleventh postoperative day.

On the ninth postoperative day, there was considerable abdominal distention despite good action of the Levine tube. On that day, a jejunostomy was done because intestinal obstruction was suspected. At operation, marked dilatation of the jejunum was found. Following this, the abdomen became soft. Despite large amounts of daily Ringer's solution, the

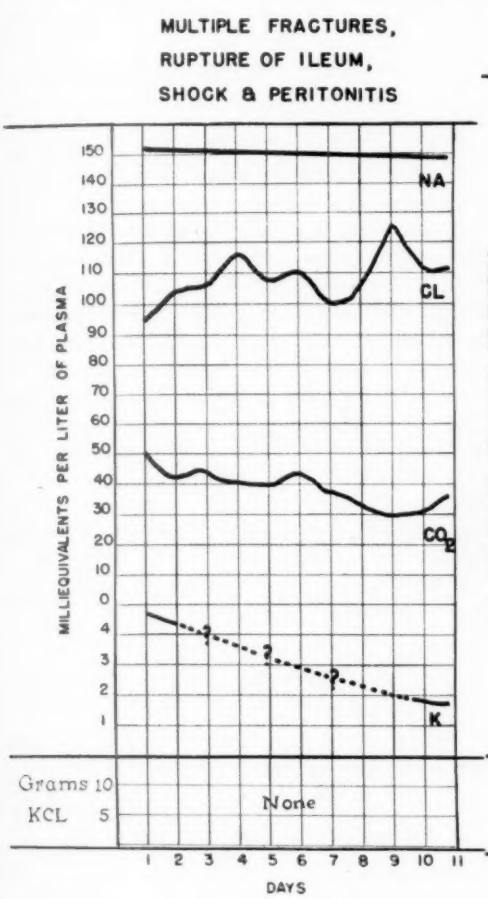

FIG. 7

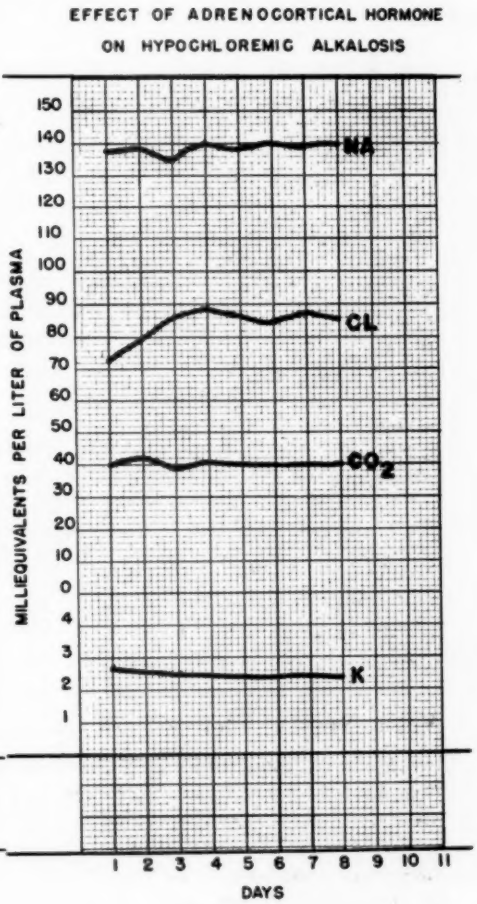

FIG. 8

blood NPN and CO_2 remained elevated. On succeeding days his condition was critical, although urine output was good. On the last day of life the patient appeared critically ill, with marked muscle weakness. A serum potassium determination was carried out shortly before death, too late, however, for potassium therapy to have been instituted after the finding of a plasma level of potassium of 1.76 MEQ. On the last five days of life a satisfactory urine output (about 1 liter per day) was maintained.

Comment. Figure 7 illustrates the blood chemistry findings on this patient. Despite large amounts of Ringer's solution given daily, with plasma chloride

levels appearing fairly normal throughout, a persistent severe alkalosis was present. This can be explained by the findings of extremely low potassium level on the day of death. In retrospect, it is realized that the patient probably was losing around 100 MEQ of potassium per day; this loss was in no way corrected by the large amount of Ringer's solution given. In patients who have experienced severe trauma, and in whom several surgical operations must be performed, urinary loss of potassium may reach high levels because of adrenal sympathetic overactivity.

IX. Effect of Adrenal Cortical Hormone on Hypochloremic Alkalosis (Case Report).

L.C. (B46135). This 36-year-old woman entered St. Philip Hospital May 5, 1948, after having been in coma for 2 days, which was preceded by bowel and urinary incontinence. A ventriculogram on the day of admission showed a large left frontal cyst in the cerebrum. Craniotomy this same day revealed by study of a biopsy an unusual malignant tumor of blood vessel origin, probably an hemangioblastoma. In the immediate postoperative period, severe hypotension (blood pressure 80/50) developed, and was treated by large amounts of saline solutions, and adrenal cortical extract (22 cc. first day) and neosynephrine. Blood chemistry findings on the first postoperative day were K 2.7 MEQ, Na 138 MEQ, chloride 73 MEQ. During the first 2 postoperative days 4000 cc. saline and 1000 cc. whole blood were administered, 650 cc. urine being excreted. On the third postoperative day, 3000 cc. saline, 2000 cc. 10 per cent glucose in water, and 85 cc. adrenal cortical extract were administered; this was followed by a urine output of 1850 cc. Adrenal cortical extract (20 to 40 cc.) and 2-3000 cc. saline solution were given on the fourth and fifth postoperative days, with return of blood pressure to normal levels, and satisfactory urine output (750 and 1480 cc.). Figure 8 indicates, however, that despite these large amounts of saline solutions, alkalosis and hypochloremia were never corrected. Blood NPN never rose above 38 mgm. per cent. The patient succumbed on the eighth postoperative day, having never recovered from coma.

Comment. The chemical observations on this patient substantially corroborate our impressions of the effect of adrenal cortical hormone in the first patient studied who received large amounts of this hormone and in whom likewise alkalosis and hypochloremia failed to be corrected by the administration of massive amounts of saline. In the first patient, plasma sodium levels rose to 171 and 176 MEQ; whereas in the patient under discussion, plasma sodium concentration stayed within normal limits.

Internists have been aware for some time of the influence of adrenal cortical hormone in promoting urinary excretion of potassium and suppressing bicarbonate and sodium excretion. It remains for surgeons to recognize the possible dangers attendant on the use of adrenal cortical hormone in the therapy of traumatic and surgical shock. In our clinic, we have largely abandoned the use of adrenal cortical hormone for shock therapy because (1) it is more than likely that accidental or surgical trauma sets in operation the discharge of adrenal cortical hormone in sufficient amounts to effect renal tubular function, and (2) this hormone has been found relatively ineffective in raising the blood pressure of patients in shock.

THE MANAGEMENT OF POTASSIUM DEFICIENCY

In treating patients for potassium deficiency, two matters are of paramount importance: (1) how severe is the deficiency, and must replacement be rapid or permitted to be slow, and (2) the ionic composition of the replacement fluids.

Although Ringer's solution is ideal for physiologic experiments and theoretically might be suitable for replacement of fluid loss, a comparison of its chemical composition with that of gastric and intestinal fluids shows that Ringer's solution may be quite deficient in potassium. Two years' experience with Ringer's solution as a replacement fluid confirms this impression; in practice, even if used in large amounts daily, it does not sustain normal plasma potassium levels with continued loss of body fluids, and is wholly unsuitable for use as a replacement fluid in the treatment of potassium deficiency.

Several suggestions have been made for the composition of potassium replacement fluids. The chief concern of all who have studied this problem has been the danger of infusing potassium salts so rapidly that dangerously high levels of blood potassium would be reached and potassium poisoning result. This danger should not be minimized, although probably it is not great when severe potassium depletion exists. Darrow[10] advises the infusion of 0.26 Gm. KCl per Kg., over a period of four to eight hours; Bellet[11] uses isotonic KCl solutions (1.14 Gm. KCl per 100 cc. of water), giving 100 to 700 cc. in one to three hours; at the Presbyterian Hospital, N. Y., Dr. Lockwood's group[7] uses two solutions (1) 2.33 Gm. KCl + 6.44 Gm. NaCl per liter and, (2) where sodium is not required, 2.33 Gm. KCl in 1 liter 5 per cent glucose in water. So far as we can determine, there need be no fixed rule for the composition of the KCl solutions; more important is their intelligent use.

The emergency treatment of severe potassium deficiency requires considerable care and a nicety of clinical and chemical judgment, because the patient's condition (extreme muscular weakness, etc.) may demand the relatively rapid infusion of large amounts of potassium chloride in a one to two-hour period. If the state of renal function is not known, KCl infusions should be preceded by the intravenous infusion of 600 to 800 cc. 5 per cent glucose in water to stimulate urine flow. A pre-treatment estimation of plasma potassium is helpful; and if the potassium level is low (below 3.0 MEQ), relatively rapid rates of potassium administration are less hazardous. Serial electrocardiograms before and during potassium administration may be used to guide rapid potassium therapy (see Bellet). Our experience indicates that the important thing is to give the first few grams of KCl over a period of one to four hours; this usually takes care of the emergency phase of potassium depletion. From then on replacement may be less rapid, and indeed leisurely, care being taken to continue therapy (2 to 4 Gm. of KCl per day) intravenously, *above* daily losses. Once plasma bicarbonate content has fallen to normal levels after alkalosis, one can usually conclude "the potassium situation is well in hand."

Oral administration of KCl should be begun as soon as possible. It makes little difference what type of solution is used just so it does not induce nausea. We regularly give 5 to 15 grains KCl in tablets by mouth or in an ounce of water put through an indwelling gastric or jejunostomy tube. In some patients in whom muscle biopsy analysis has demonstrated severe depletion, we have given larger amounts of potassium chloride (up to 5 Gm.) by mouth or tube. These amounts are usually given every three to four hours over a 24-hour period. If the patient is taking food by mouth, potassium intake may likewise be supplemented with oral KCl.

From what has been demonstrated above in the charts of the clinical cases, it follows there is a most important preventative phase to the whole potassium deficiency problem. The restriction of gastric and intestinal drainage to the shortest possible time, adequate daily replacement of sodium and potassium chloride above daily losses before and after operation, the restriction of intravenous fluid therapy to what is actually required after intelligent appraisal of the individual patient's need (rather than adherence to a set clinical rule of fluid and salt replacement), all, if carried out, make easier the surgeon's problem. Finally, early withdrawal of "all tubes from all orifices," allowing ingestion of well-cooked, appetizing food even in small amounts* and observation of what Francis Moore calls "the need to be left alone," will diminish greatly the number of patients who suffer from this deficiency.

Potassium deficiency is one of the complications that has followed the extension and increased complexity of surgical care for what were once patients without hope. We have no way now of knowing how many surgical patients suffer from this deficiency, but when we consider how common is the use of the Wangensteen tube, we can suspect the number to be fairly large. Recognition and management of this deficiency is relatively simple when thought is given to the manner in which it may be produced.

SUMMARY

A brief consideration is given of the common causes of potassium deficiency in surgical patients. Case reports with accompanying charts are presented illustrating the points to be remembered in the recognition and management of potassium deficiency.

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A lucid description of the mechanism by which potassium deficiency is produced is to be found in the October, 1949 American Journal of Medicine by T. S. Danowski. (Vol. 6, p. 525.)

TECHNIC OF ANASTOMOSIS OF THE BRANCHES OF THE FACIAL NERVE WITH THE SPINAL ACCESSORY FOR FACIAL PARALYSIS*

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THE CHOICE OF OPERATION for facial paralysis is generally determined by the location of the lesion which causes the paralysis. If the nerve is destroyed intracranially, an anastomosis of the facial with some other cranial nerve must be done to restore motor function. In lesions of the nerve in the bony canal, an anastomosis with the hypoglossal or spinal accessory has often been done. However, Ballance and Duel,[1] some years ago, advocated exposing the nerve in the facial canal and using a nerve graft for filling the defect. Extracranial injuries of the nerve may often be sutured directly, whether the lesion is in the trunk or in one or more important branches.

In the experience of the writers, there has been no return of normal emotional expression following anastomosis or suture of the trunk of the facial nerve. Lack of emotional expression has also been observed following recovery of motion from a severe Bell's palsy. The facial nerve has a high capacity for recovery of motor function, but this is not equivalent to recovery of complete function following lesions of the trunk, inasmuch as emotional expression is practically completely abolished. Moreover, in nearly all cases following direct suture, anastomosis, or recovery following severe Bell's palsy, the final results are marred by the development of mass movements. For instance, when a patient closes his eye on the previously paralyzed side, it is likely to bring into activity the musculature of the entire side of the face. Ford and Woodhall[2] made a study of this phenomenon some years ago and came to the conclusion that mass movements were due to straying fibers, so that down-growing fibers from the proximal trunk, intended only for one branch, might go into all three branches, producing a functional unit incapable of the dissociated action so essential to facial expression. We believe this is the best explanation yet advanced for mass movements.

We have observed that objectionable mass movements do not develop following recovery from suture of individual branches. Results of surgery in these cases are much more complete and satisfactory than when the trunk has been divided.

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949.

A recent case of facial paralysis was very instructive in regard to the type of recovery which followed operation. The treatment in this case led to an investigation of the anatomy of the spinal accessory and hypoglossal nerves. Information obtained from this study explains several matters that heretofore have been very much confused so far as the writers were concerned.

Case Report. The patient in this case was a 14-year-old girl, who was first seen on September 3, 1948, with a complete left facial paralysis. A parotid tumor had been

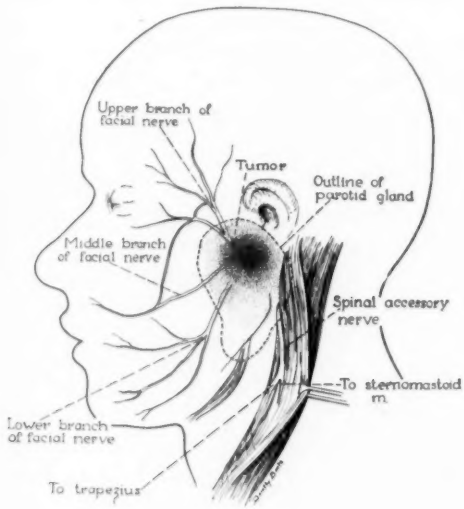

FIG. 1

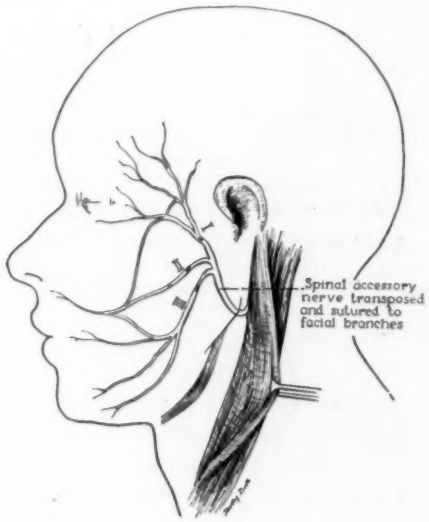

FIG. 2

FIG. 1.—The stippled area in the sketch indicates the location of the parotid tumor, involving the branches and the trunk of the facial nerve. Outline of the parotid gland is shown by a broken line. The trunk and a portion of the branches of the facial nerve had been excised, along with removal of the tumor, and it was impossible to use the trunk of the hypoglossal in anastomosis with the peripheral branches of the facial. The spinal accessory is shown, with one large branch going into the sternomastoid muscle and continuing on into the trapezius. This is probably the most frequent anatomical arrangement of the spinal accessory.

FIG. 2.—The branch of the spinal accessory to the sternomastoid muscle and the trunk going to the trapezius are divided. By dissecting the branch from the trunk for about 2 cm. and following a line of cleavage in the trunk itself for the same distance, it was possible to fashion three prongs on the end of the spinal accessory. These were sutured, as indicated, to the three main branches of the facial, using one arterial silk suture.

removed 5 years before, but recurred, and a more extensive operation was performed in another clinic 5 months previously, followed by left facial paralysis. The tumor was said to have been a benign, mixed type.

Examination of the patient 5 months after the onset of the paralysis showed no evidence whatever of return of function. Moreover, there was complete reaction of degeneration of the paralyzed facial muscles. It was decided to explore the region of the left parotid, hoping to find it possible either to do a direct suture of the facial nerve or to do an anastomosis with the hypoglossal or spinal accessory. After a very long and tedious dissection through scar tissue at the site of the removal of tumor, it was found that the nerve had been destroyed from a point proximal to the pes anserina and

distally to the level of the anterior border of the parotid gland. We were unable to locate the proximal trunk in the dense scar tissue of the retromandibular fossa. It was found impossible to approximate the separate branches of the facial nerve to form a bundle for suture into a substitute nerve. Because the procedure had been quite long and the operator was not sure just what he should do, it was decided to postpone further efforts at that time. A loose black silk ligature was placed on each branch of the facial nerve for later identification. The spinal accessory and the hypoglossal were exposed and similar identifying ligatures were used.

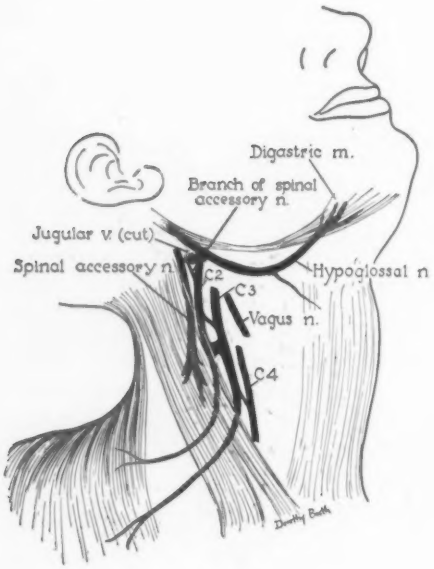

FIG. 3

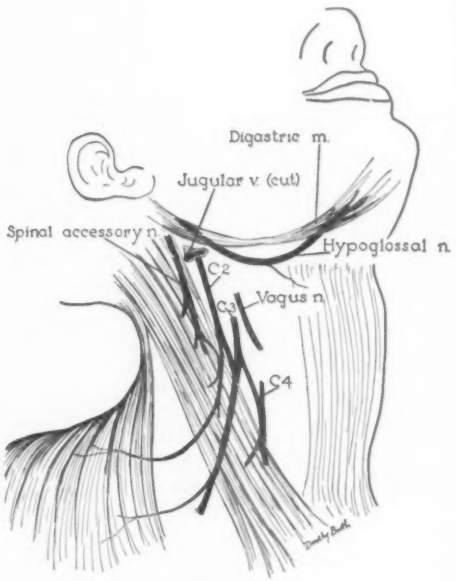

FIG. 4

FIG. 3.—Diagrammatic sketch of a dissection. The spinal accessory nerve divides at its exit from the jugular foramen. A large branch courses in front of the jugular vein to supply the sternomastoid muscle. A much smaller branch passes behind the jugular vein to follow the cervical branches to the trapezius. The large terminal branches of the hypoglossal are shown as they pass through the hypoglossus muscle to the intrinsic muscles of the tongue.

FIG. 4.—The spinal accessory nerve is shown here supplying the sternomastoid muscle, but with few, if any, fibers going to the trapezius. One branch was traced down to the branches of the second cervical nerve, but could be easily separated from it.

In similar cases, prior to the one being discussed, we had used fascial strips to support the paralyzed side. This operation in the hands of Brown[3] had given good results in restoring balance to the face in repose. However, we have never been satisfied with the fascial strip operation and have much preferred using some method that would bring about re-innervation of the facial muscles if this were possible.

Six days after the initial procedure, the wound was reopened. In the meantime, it had been decided to split either the spinal accessory or the hypoglossal to form three branches which might be separated sufficiently to bring about approximation with the branches of the facial. We then investigated the spinal accessory. It was traced under the sternomastoid and one large branch was found to enter this muscle. This branch was divided as deep in the muscle as possible. The main trunk of the spinal accessory was then divided, giving a segment the same length as the branch to the sternomastoid.

The branch was separated from the trunk for a distance of 2½ cm. This was surprisingly easy and did not injure the main trunk. It was then found that the trunk in this case had two other components which could be separated, giving two segments the same length as that of the branch to the sternomastoid. Thus, there were three branches of the spinal accessory to be sutured to the distal branches of the facial. Approximation was carried out, using one through-and-through arterial silk suture for each nerve. The wound was closed in layers and the patient was returned to her room in good condition. She left the hospital 8 days after the second operation with the wound well healed.

The patient returned for check-up examination on January 6, 1949, 3 months from the date of operation. There was good movement in the angle of the mouth and she could close her eye weakly but completely on the previously paralyzed side. Recovery had been much more rapid than in the usual case following anastomosis, and we attributed this to the fact that suture was made much more anteriorly than in the average case.

Examination 6 months after operation showed the face well balanced, there were no mass movements and practically no evidence of atrophy around the left shoulder. She had some difficulty immediately after operation in elevating the left arm unless it was first brought forward. The absence of associated movements was most gratifying. The patient could elevate her shoulder without its affecting the facial muscles. The sternomastoid was completely paralyzed in the upper part, but the lower fourth seemed to have some contraction. This was thought due to an independent nerve supply from the cervicals.

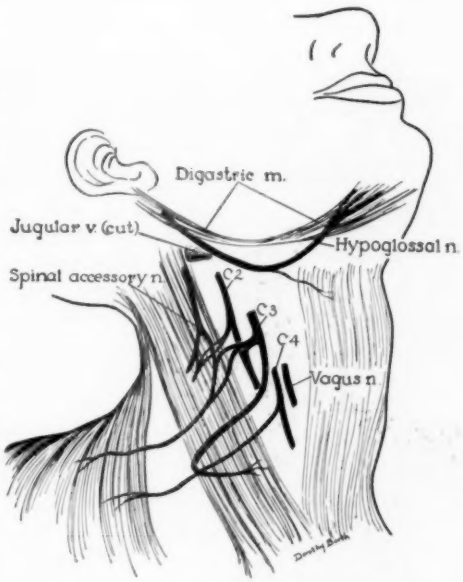

FIG. 5.—Another variation of the spinal accessory nerve is shown. Here a branch of this nerve splits a branch of the second cervical nerve and courses on to join the third cervical nerve branch to supply the trapezius. Note the large branch of the third cervical passing through the sternomastoid, then curving back to end in this muscle.

This patient was a most satisfactory candidate for treatment of facial paralysis. She had a naturally subdued expression, rather a "poker-face" type, with little vivacity. She was advised not to try re-education of the previously paralyzed muscles before a mirror as this, in our experience, only makes matters worse.

In this case the spinal accessory was used because it was better suited anatomically. If the hypoglossal were used it would have been necessary to trace the nerve into the hyoglossus muscle, where three or four large terminal branches are usually found.

Following the first operation on this patient we decided to do some anatomic investigation in an effort to explain the variable results of surgeons who have used the spinal accessory in anastomosis rather than the hypoglossal. We found the spinal accessory nerve is most inconstant in its anatomy. It is

smaller than the hypoglossal. Some of the anomalies found in dissection of this nerve will be illustrated.

In a paper[4] on facial paralysis read before this Association by one of us (C. C. C.) in December, 1944, we expressed the opinion that the use of the hypoglossal in anastomosis with the facial nerve caused practically no disability, while section of the spinal accessory did often carry considerable disability. Woodhall, in a personal communication about this time, stated that "paralysis" of the spinal accessory was incompatible with "full military duty."

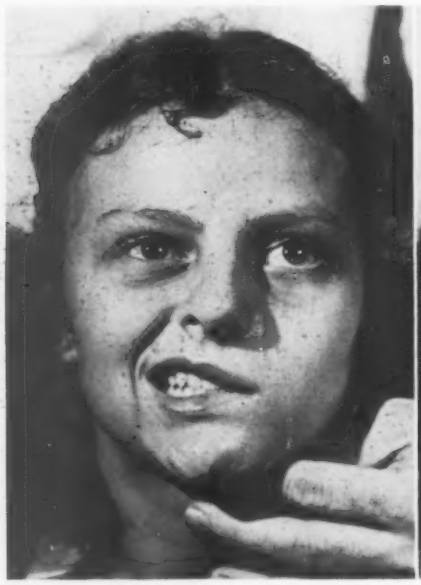

FIG. 6

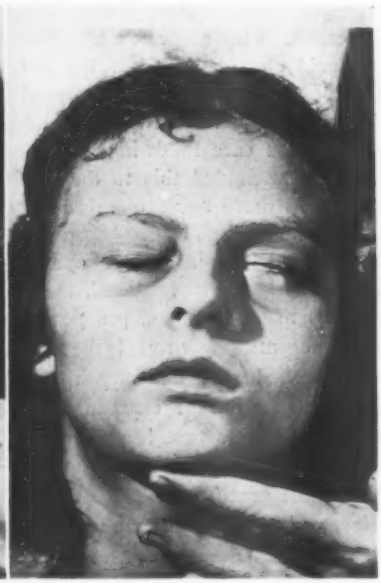

FIG. 7

FIG. 6.—Photograph of patient before operation making an effort to show her teeth. The left side does not move at all but the right is in firm retraction.

FIG. 7.—Photograph made before operation with the patient trying to close her eyes. It is noted the patient has considerable narrowing of the palpebral fissure due to relaxation of the levator supplied by the third nerve. This patient shows more ability to close the eye than most but still there is complete left facial paralysis.

However, we now believe that in some cases following section of the spinal accessory nerve, there may be real disability while in others the disability is negligible and we believe this variation is due to variation in the anatomy of the nerve. In some cases the spinal accessory does not extend any further than the sternomastoid muscle and the entire supply of the trapezius is given off by the cervical nerves. In other cases, the spinal accessory divides higher up, one branch passing in front of the internal jugular vein to enter and end in the sternomastoid, and the other coursing behind the jugular vein to supply the trapezius. If only the most accessible branch is divided, that is, the anterior

one supplying the sternomastoid, obviously the patient would have no trouble with the shoulder.

In the case here reported we have also been impressed by the fact that mass movements did not develop. There may be an anatomical explanation for this. The three peripheral branches of the facial nerve were sutured to the three prongs of the spinal accessory made by using the branch to the sternomastoid and by separating the main trunk. Therefore, this procedure was very similar to direct suture of facial nerve branches and may account for the absence of mass movements. For instance, impulses intended for the orbicularis muscle

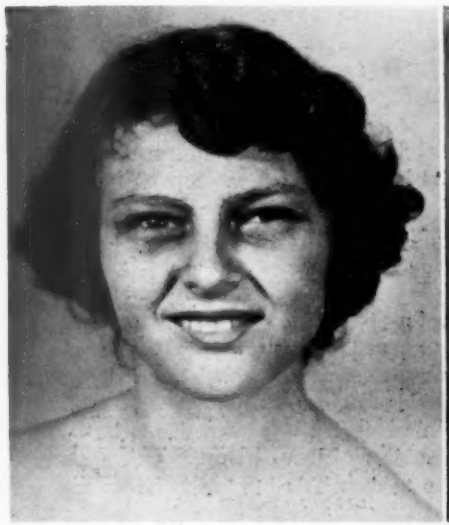

FIG. 8

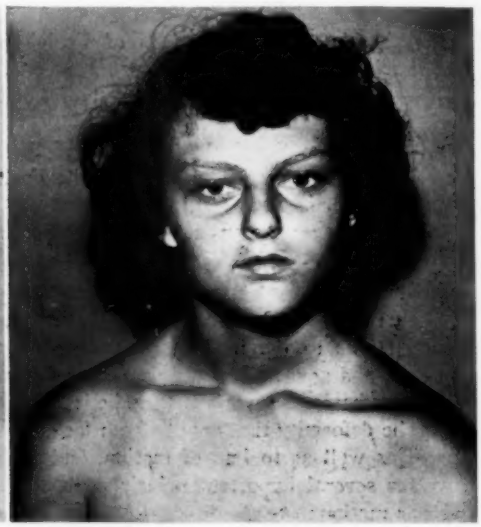

FIG. 9

FIG. 8.—Photograph of patient one year after facio-spinal accessory anastomosis. The face is well balanced, the palpebral fissure is of normal width and there is no evidence of paralysis.

FIG. 9.—Photograph one year after operation. Note the absence of atrophy about the shoulder. There is often severe atrophy and disability of the shoulder following section of the spinal accessory but this depends on the anatomy of the nerve, which may vary considerably.

would not be transmitted by straying fibers into other domains of the facial nerve. If this impression is sound and mass movements are due to misdirected fibers, then it may be inadvisable at any time to do a trunk-to-trunk anastomosis, if the method herein described is applicable.

The results of surgery for facial paralysis are by no means perfect but great improvement can be obtained by restoring motility to the paralyzed muscles, thus bringing about facial symmetry, protection to the eye, and the prevention of atrophy. The inability to restore normal emotional expression is the greatest obstacle to satisfactory results of surgical treatment of facial paralysis. Loss of normal emotional expression may also be a permanent residual of a severe Bell's palsy. This lack of recovery, apparently being due

to faulty regeneration of the nerve, very probably will remain an insurmountable obstacle. It would seem from the case herein reported that there may be some likelihood of overcoming the other two problems, mass movements and associated movements, which mar the success of surgery of the facial nerve.

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Discussion—Dr. Frank H. Mayfield, Cincinnati: Dr. Coleman has made many contributions to the field of neurologic surgery, and in particular to peripheral nerve surgery, and now he has added another fundamental one. It is stimulating indeed to note that after beginning work in 1904, he is still making original contributions.

The deformity that results from facial paralysis is in a large part due to lack of symmetry as well as to loss of motion, and the subject matter of Doctor Coleman's paper involves several important principles that have to do with restoring facial symmetry as well as motion:

First, the demonstration from dissections in the cadaver that there are many anatomic variations in the spinal accessory nerves, and that this nerve can be utilized in certain instances without producing associated shoulder disability of any degree.

Second, it utilizes the principle of bringing viable nerve tissue close to its ultimate destination, hence reducing the distance that the nerve has to grow before function can return. The extremely rapid recovery noted in this case must be due to the fact that the nerve had to grow a very short distance.

Third, he has accomplished restoration of function without associated movements and without massed movements. There are several possibilities that one must consider in order to explain the associated and massed movements that occur after facial paralysis. One is that the nerve which one transplants has a different cortical representation from the facial nerve; another explanation is, as Doctor Coleman commented, that the nerve fibers stray. Actually they do more than stray; they split and develop into forks, with two or more branches from a single axis cylinder. It is readily seen that these branches could split into different distal trunks and thereby incorporate many functional units of the face into one. If, however, one transplanted the nerve distal to the branches, the chances of error of this sort would be reduced.

Another possibility to explain the associated movements is that there is an actual transfer of impulses at the point of suture from one nerve bundle into another as the result of lack of maturation of the fibers. The chances of large functional units being incorporated into one on this basis would also be lessened if the transplant were made into smaller nerves that comprise the more distal branches. The possibilities of this are so stimulating that I would like to ask Doctor Coleman if he would by election use this procedure in

a patient with facial paralysis in whom it was possible to identify the main trunk of the facial nerve. Since he has shown that it is possible to divide the ends of the spinal accessory and that it is also feasible to dissect out the distal branches of the facial nerve, would he by election plan to make the transplant distal to the pes anserunis?

It is fortunate, in reference to facial nerve surgery and the developments which this case yields, that the patient was operated upon elsewhere than in Doctor Coleman's clinic for the parotid tumor, for had it been done there, there would have been no facial paralysis in the first place. It should be possible to remove the parotid gland from the facial nerve without paralyzing the nerve, even when the lesion of the parotid is malignant.

The operation which Doctor Coleman has described will be extremely useful in many cases of facial paralysis, but paralysis secondary to surgery for a parotid gland tumor should not be frequent.

In closing, I would like to take this opportunity to thank Doctor Coleman and the Southern Surgical Association for the privilege of attending this most delightful meeting.

DR. JOSEPH E. J. KING, New York: If any ordinary doctor had told me about this result obtained by Doctor Coleman I would not have believed it even if I had seen the pictures. However, I believe every word he said. It is a most unique case and the result is most astounding. I have never seen such complete return of function following any type of operation on or pertaining to the facial nerve. So far as I know, this is the first time such an operation has been performed; at least I have never heard of any such thing. It is just like him to have figured out such an operation in his own mind.

Everyone is acquainted with anastomosis of the spinal accessory and the hypoglossal nerve to the distal end of the facial, and we have obtained pretty fair results from these procedures. I do not think it makes any difference which of the nerves is used except that the spinal accessory nerve would be the one of choice in an individual who is in a public position—like a cashier in a bank—because of the ensuing hemi-atrophy of the tongue following the use of the hypoglossal nerve. In addition, almost everyone is acquainted with the sling or facial slips as used by Barrett Brown, also the nerve graft that was so ably used by Thomas Tickle, laid along the facial canal in the mastoid portion of the temporal bone where that portion of the nerve is destroyed.

Not only is Doctor Coleman to be commended for the unusual use he has made of the spinal accessory nerve, but also for overcoming the technical difficulty of suturing the small portions of the spinal accessory nerve to the even smaller three ends of the distal portion of the facial nerve. It is almost like suturing a spider web.

I feel sure no one has ever seen pictures showing a better result than that obtained by Doctor Coleman in this case, and certainly not in the short time which elapsed before return of function occurred. The individual action, instead of the mass action of the muscles, is also commendable. He deserves a lot of credit.

When I recall how hard he worked in the First World War in the Neurosurgical Services in Cape May and Fox Hills, spending three or four hours at a time in the tedious repair of peripheral nerves and wearing himself out at these repeated tasks, I should think that by this time he would be taking things more easily, like the rest of us. Instead of that he keeps on working just the same. I offer my sincere congratulations.

DR. CLAUDE C. COLEMAN, Richmond, Va. (closing): I want to thank Doctor King and Doctor Mayfield for their discussion. They are good friends of mine and I think this accounts for the complimentary remarks concerning my paper.

There is one very pertinent question Doctor Mayfield asked. That was whether I would be willing to use this method of anastomosis rather than use the trunk of the nerve, which has been the custom heretofore. Of course, I have had experience with only one case and, while I am not in position to settle the problems of anastomosis on this one case, I do think that if I could find a good donor nerve in which the branches could be used satisfactorily, I would be inclined to choose this method in preference to using

the trunk. The choice of nerve for anastomosis in these cases would depend largely on the anatomy of the donor nerve. If the nerve one wishes to use to innervate the paralyzed facial branches cannot be split into components to supply each branch, then this nerve will have to be rejected.

Doctor Mayfield had a method of suturing the trunk of the donor nerve into a number of branches but I understand this is possible only when the branches can be brought together in a bundle. This is not often possible with the facial.

In the study of anatomic specimens it is found that the hypoglossal nerve is rather constant while the pattern of the spinal accessory is most inconstant. If the hypoglossal is traced into the hyoglossus muscle where it enters the intrinsic muscles of the tongue, three or four good sized branches will be found. The hypoglossal is larger than the spinal accessory. In examining the components of the nerve plexus in the dural canal, one is impressed with the increase in size of these nerves extraspinally.

The case reported was a rather interesting one, particularly from the standpoint of anatomy.

BOOK REVIEW

Operating Room Technique, by Edythe Louise Alexander. C. V. Mosby Co., St. Louis, Mo., 1949.

This is the second edition of a volume which, since its introduction in 1944, has become a standard reference work for operating room supervisors and nursing instructors. The book presents in a simple, well-illustrated style the essential features in the operation and management of an operating room. The chapters dealing with nursing personnel, sterilization, standardization of duties, instruments, needles and sutures are excellent and no doubt are largely responsible for its popularity with operating room personnel. Some obsolete material has been carried over into this edition however. The section on blood transfusion is devoted almost entirely to methods of direct transfusion or the open method of indirect transfusion. With commercially prepared vacuum bottles so readily available such material has little more than historical value. This however in no wise detracts from the usefulness of the book and it should prove invaluable to operating room nurses.

Oscar Creech, M.D.

NON-CONTUSIVE RADICAL MASTECTOMY*

WILLIAM F. MACFEE, M.D.

NEW YORK, N. Y.

THE THEORETICAL DANGER of disseminating cancer cells by the manipulations incident to radical mastectomy has long been recognized. Both Halsted[1] and Meyer[2] indicated in their original articles an appreciation of this possibility. The experimental studies made with animal tumors by Tyzzer[3] and by Knox[4] have provided strong evidence that the danger of producing metastases in this way is real.

In human cancer it is obviously not possible to appraise correctly all the factors that contribute to the production of metastases. Among clinicians, however, there is a widespread conviction, based upon experience, that unnecessary handling of the cancerous breast is to be avoided. In actual practice it must be admitted that this injunction is often ignored.

In 1885 Arpad Gerster[5] wrote as follows: "The handlings or manipulations ordinarily in use during the performance of bloody operations on tumors to be dissected out of surrounding tissues have positively the character of massage, and occasionally a very rough form of massage, too; and the employment of anesthetics has certainly not had the effect of increasing the gentleness of operative interference. Suffice it to say that the manipulation employed on a tumor of the breast and the lymphatic glands occupying the adjacent axillary cavity, during an operation lasting one or two hours, certainly may have the dignity of a manipulative seance." It can hardly be said that 64 years of surgical progress have entirely erased this indictment.

At best, radical mastectomy is an extensive operation, and involves an undesirable amount of manual and instrumental handling. It is possible, however, to dispense with a large part of the manual and instrumental insults to the breast by substituting the force of gravity for purposes of retraction. The utilization of gravity is effected by postural changes of the patient co-ordinated with a definite plan of operative procedure. The plan of procedure to be described involves a primary lateral approach, and, with the exception of the postural changes, is essentially that employed by the late George H. Semken.[6] The steps in the co-ordinated plan are divided into two phases.

First Phase. The patient is turned on the side opposite the lesion until the affected breast tends to fall toward the unaffected side (Fig. 1). Sand bags or other suitable supports are used to maintain this position. The skin incisions are then lightly outlined with the knife. The lateral flap of skin and subcutaneous fat is raised and extended until the anterior border of the latissimus dorsi muscle has been reached. This muscle is exposed from its tendinous portion above to its origin below. During this part of the dissection, the long subscapular nerve and accompanying vessels are uncovered at their points of entry into the muscle and cleared to the level of their disappearance in the lower

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949.

axillary fat (Fig. 2). Thorough exposure of the distal portions of the nerve and vessels at this stage greatly facilitates their identification and preservation during the axillary dissection which is conducted later from above.

The dissection is then carried medially across the space between the latissimus dorsi muscle and the lateral chest wall. This area is completely cleared from its lower angle upward to the axilla proper and its contents are reflected upon the lateral chest wall. The dissection is continued, uncovering the serratus anticus muscle to the approximate level of the long thoracic nerve. This completes the first phase, or the lateral portion of the dissection. The exposed surfaces are covered with protective moist gauze pads.

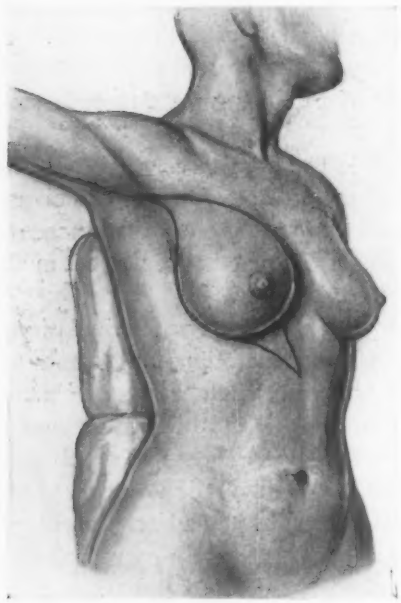


FIG. 1.—For the first phase of the operation the patient, lying on the horizontal table, is turned on the side opposite the lesion until the affected breast hangs toward the unaffected side. The incision is then outlined as shown in the diagram. It will be observed that the axillary portion of the incision is carried posterior and inferior to the axilla instead of being placed in the usual superior and anterior position. This permits thorough dissection of the axillary contents but leaves the resulting scar in a somewhat concealed position.

Second Phase. The sand bags or other supports that have been used to maintain the rotated position of the patient are now removed and the patient lies flat on the back. A small bag is left under the shoulder to bring the axilla forward. The head of the table is then elevated to an angle of about 45 degrees from the horizontal (Fig. 3A). In this position the breast tends to fall downward and laterally, that it, away from the mid-line and away from the axilla.

The medial flap of skin, with a thin layer of subcutaneous fat, is elevated and the undermining is continued until the extent of the tissues to be removed has been reached. The limits of exposure above are the humeral insertion of the pectoralis major muscle, the anterior border of the deltoid muscle with the cephalic vein, and the clavicular origin of the pectoralis major muscle. The medial boundary begins at the episternal notch and extends downward along the septum between the sheaths of the recti abdomini muscles.

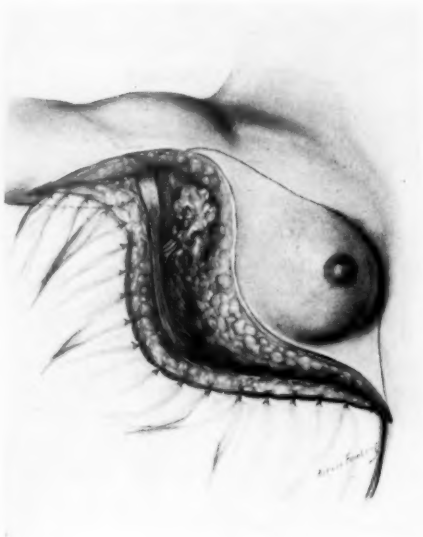
The pectoralis major muscle is then detached, first, at its humeral insertion, and then from its clavicular, sternal, and costochondrial origins. This completely releases the principal muscle by which the breast is held to the thoracic wall and the whole mass begins to fall away from the chest and axilla (Fig. 3).

The axillary dissection is then begun just above the cephalic vein and carried downward over the axillary vein, which may be conveniently and safely exposed at the point where it passes under the clavicle. Cutting the insertion of the pectoralis minor muscle removes the last important support of the breast, and the axillary contents, as they are dissected out, fall away with the breast (Fig. 4). Identification of the subscapular nerve and vessels is greatly facilitated by their previous exposure during the first phase of the operation.

After the axillary dissection has been completed the remaining fascial and muscular attachments which hold the mass are freed and the breast gradually slides off the chest wall (Fig. 5). The removal of the breast is thus accomplished in a definite orderly manner with a minimum of handling.

The wound is closed with interrupted silk sutures. Skin grafting is done if necessary. Retention sutures are not recommended. When their use is necessary for closure, there is too much tension, and sloughing of the flaps with its unpleasant consequences is likely to occur.

Between 1925 and 1941 the plan of procedure outlined above has been carried out, at least in principle, in 111 consecutive cases of carcinoma of the



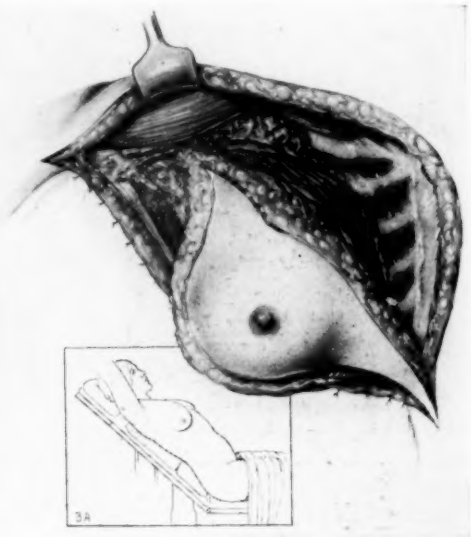


FIG. 2 FIG. 3

FIG. 2.—While the patient remains turned toward the unaffected side, the lateral incision is gradually extended in depth until the anterior margin, tendon, and medial surface of the latissimus dorsi muscle are uncovered. The distal ends of the subscapular nerve and vessels shown in the diagram are exposed at their points of entry into the latissimus dorsi and dissected free to the level of the lower axilla. The dissection of the lower axilla and the area between the medial surface of the latissimus dorsi and the chest wall is now carried anteriorly, exposing the serratus muscle almost to the level of the long thoracic nerve. This completes the first, or lateral phase of the operation. Throughout this phase the force of gravity pulls the breast away from the line of dissection and little or no manual retraction is necessary.

FIG. 3.—The supports which kept the patient rotated toward the unaffected side during the first phase of the operation are now removed, allowing a return to the full recumbent position. The head of the table is then elevated to an angle of about 45 degrees as shown in the insert (3A). As the dissection proceeds, the position of the patient causes the breast to fall downward and laterally away from the axilla and the anterior chest wall. Artificial retraction is not necessary.

breast, operated upon personally, or assisting and supervising a resident surgeon. Almost exactly half of the patients were from the ward service and the remainder were private and semiprivate. The ages ranged from 28 to 80 years, the average being 54 years. Thirty-six patients, or 32.4 per cent, were without axillary lymph node metastasis, 75 patients, or 67.6 per cent, had axillary node involvement.

The group of cases represents approximately 90 per cent of those presenting themselves for treatment. It includes some who were chronically ill with other diseases and some in whom a cure could not reasonably be anticipated. In some instances it might have been postulated that the patient would die of some intercurrent condition but it has been my observation that whatever else the patient may have it is likely to be the cancer to which she finally succumbs.

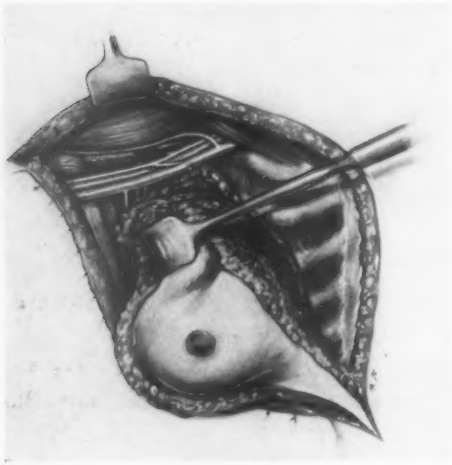


FIG. 4

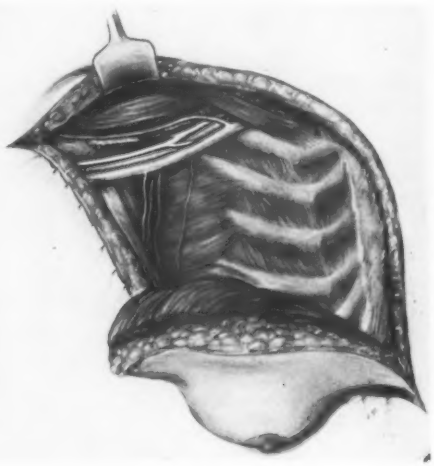


FIG. 5

FIG. 4.—Dissection of the axilla is continued and the tissues fall away from the vessels and nerves. The retractor shown in the diagram is used temporarily to deflect the axillary contents forward, while dissection about the subscapular nerve and vessels is completed. The narrow attachment of tissue remaining at the lower angle of the incision assists in directing the rotation of the breast downward and outward as it is freed above.

FIG. 5.—The amputation is now virtually completed, with the breast, as its attachments are severed, falling away of its own weight from the chest wall.

Another good reason for operating upon patients for whom a cure cannot be expected is the fact that radical mastectomy is one of our most effective palliative measures, particularly with reference to the regional manifestations of the disease. If one can relieve or spare the patient of local ulceration and pain due to compression of the brachial plexus by involved lymph nodes, a worthwhile service is performed, even though the sufferer eventually dies of hidden metastatic lesions.

Haagensen,[7] in discussing the curative value of radiation, has stated that the high hopes entertained a few years ago have not been fulfilled and that we must turn back to surgery. It is likewise true that radiation as a palliative measure has its limitations. It is useless in ulcerating lesions, and when applied to relieve the pressure of a carcinomatous mass on the brachial plexus, the last state of the patient, as the late Dr. Frank S. Mathews was wont to say,

is often made worse than the first. The fibrosis due to cancer is merely increased by that due to irradiation.

The principal criterion for selection of the case for operation in this series was simply whether it appeared possible to encompass all of the regional tumor within the operative field. Such conditions as advanced age of the

TABLE I.—*Results of Radical Mastectomy in 36 Cases of Carcinoma of the Breast Without Axillary Lymph Node Involvement.*

	Five Years		Ten Years	
	No.	Per Cent	No.	Per Cent
Living and apparently well	22	61.1	11	30.5
Died of recurrence or metastasis	6	16.6	9	24.0
Living with recurrence or metastasis	1	2.5	1	2.5
Died of other causes	3	8.3	2	5.5
Postoperative deaths	2	5.5	0	0
Results unknown	2	5.5	11	30.5

patient, intercurrent disease, or ulceration of the tumor were not considered justification to withhold surgical treatment, except in extreme cases.

The results of radical mastectomy performed in the manner described are shown in Tables I, II, and III. It is evident from the tables that with this type of radical mastectomy in which a conscious effort is made to avoid contusive manipulations, the results show no significant variation from those obtained over a comparable period, and in comparable clinics with the standard

TABLE II.—*Results of Radical Mastectomy in 75 Cases of Carcinoma of the Breast With Axillary Lymph Node Involvement.*

	Five Years		Ten Years	
	No.	Per Cent	No.	Per Cent
Living and apparently well	17	22.6	9	12.0
Died of recurrence or metastasis	46	61.3	53	70.6
Living with recurrence or metastasis	5	6.6	0	0
Died of other causes	2	2.6	4	5.3
Postoperative deaths	3	4.0	0	0
Result unknown	2	2.6	6	8.0

radical procedure.[8-14] In fact, there is a striking parallelism in the results reported. Statistical data from the large private and special clinics and from general hospitals with restricted criteria of operability show somewhat higher rates of cure.[15-19]

In view of these actualities it may be seriously questioned whether the procedure that has been offered has any real merit. Obviously it could have little or no value for the patient whose tumor has already metastasized. Its theoretical and logical value is to the patient whose tumor has not metastasized but may be disseminated by careless handling. When one sees nests of cancer

cells in the lymphatics and in blood vessels it is easy to believe that they could be dislodged and become free emboli.

Before the actual worth of this or any other operative technic can be determined with accuracy, there must be greater standardization of the criteria of operability. Even so, in the interpretation of any set of rules upon which the decision for or against operation is based, it is extremely difficult to eliminate the personal element, and possibly the desire to present favorable statistics.

I am in agreement with those who believe that statistical studies of cancer of the breast should include all patients who have presented themselves for advice or treatment, and that results should be based upon the salvage from the entire group.

TABLE III.—*Combined Results of Radical Mastectomy in 111 Cases of Carcinoma of the Breast, with and without Axillary Lymph Node Involvement.*

	Five Years		Ten Years	
	No.	Per Cent	No.	Per Cent
Living and apparently well	39	35.1	20	18.0
Died of recurrence or metastasis	52	46.8	62	55.8
Living with recurrence or metastasis	6	5.4	1	0.9
Died of other causes	5	4.5	6	5.4
Postoperative deaths	5	4.5	0	0
Result unknown	4	3.6	17	15.3

SUMMARY

1. An undesirable amount of handling and compression of the diseased breast frequently occurs during radical mastectomy.

2. It has been shown experimentally that squeezing and massage of the cancerous breast in animals increases dissemination of the tumor.

3. A plan of procedure designed to minimize manipulations, compression, and possible spread of cancerous emboli during mastectomy is outlined.

4. In principle, the plan substitutes gravity for hands and instruments in controlling the positions of the breast.

5. Radical mastectomy actually becomes less complicated when this procedure is followed.

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CHEST WALL TUMORS*

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Chest wall tumors are relatively rare. It is not surprising, therefore, to find the literature on the subject consists mainly of case reports. Few authors have been primarily interested in the anatomical location of the lesion, but have mentioned the tumor in an analysis of specific types of neoplasms. For example, Campbell[1, 2] described 57 cases of osteogenic sarcoma, including one case of primary rib tumor, and also reported only one Ewing's sarcoma of the rib among 23 other cases.

Important papers considering tumors of the chest wall on a regional basis include Hedblom's[3] review of the world literature in 1933, in which he collected 291 cases and added 22 of his own. And nine years later Sommers and Major[4] added 66 examples of tumors of the bony thoracic cage which were recorded from 1933 to 1942 and also reported 15 additional cases from the University of Michigan Hospital.

Available statistical data concerning the exact incidence of thoracic wall tumors are incomplete and probably unimportant. Moreover, accurate histologic classifications of the tumors are omitted or confusing. The purpose of this communication is to indicate that the incidence of malignant tumors of the chest wall is rather high and to outline the surgical management of these lesions on the basis of their anatomical positions.

CLASSIFICATION

The types of tumors in our series are shown in Tables I, II and III. For convenience the material is presented under the divisions of benign, malignant and metastatic neoplasms.

TABLE I.—*Benign Tumors (Excluding Superficial Lipomas and Sebaceous Cysts).*

Neurofibroma	4
Fibrous dysplasia of ribs	6
Osteochondroma	3
Chondroma	4
Fibroma	2
Perineural fibroma	1
Ganglioneuroma	2
Lipomas (recurrent or deep)	6
	28

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 7, 1949.

Of the 48 primary chest wall tumors, 28 were classified as benign after microscopic examination of the tissue and 20 were found to be malignant. If the five metastatic tumors were included as malignant lesions the incidence of malignancy in the entire series would be 47 per cent.

In the cases of metastatic tumor from the kidney, the testicle and one case from the prostate, the possibility of a metastatic lesion was considered before operation. The metastatic tumor from the thyroid and in the other case of

TABLE II.—*Malignant Tumors.*

Fibrosarcoma	5
Malignant melanoma	2
Reticulum sarcoma	1
Neurofibrosarcoma	4
Unclassified sarcoma	2
Ewing's tumor	3
Hemangio-endothelioma	2
Hodgkin's disease	1
	20

prostatic carcinoma, the true nature of the tumor was not suspected before operation.

A clinical classification of chest wall tumors based on their anatomical locations was proposed by Zinninger[5] in 1930. His classification was (1) tumors originating from deep structures of the thoracic wall and having a partially intrathoracic position; (2) tumors with a superficial origin, but fixed to deep structures; and (3) those arising within the thorax and presenting through the thoracic wall.

TABLE III.—*Metastatic Chest Wall Tumors.*

Metastasis from prostate	2
Metastasis from testicle	1
Metastasis from thyroid	1
Metastasis from hypernephroma	1
	5

It seems possible that the clinical management of thoracic wall tumors may be predicated on another and less complicated classification, namely, superficial and deep.

For practical purposes superficial tumors include only those which appear to arise from the skin or subcutaneous tissue and are not fixed to the chest wall, whereas deep tumors include all other varieties. Superficial chest wall tumors are usually lipomas, sebaceous cysts, nevi and other lesions whose nature and benignity are often obvious. These have not been included in this report, a factor which, of course, increases the incidence of malignant tumors in the series.

Deep thoracic wall tumors present difficult diagnostic problems and should be considered malignant or potentially malignant neoplasms in all cases.

THE DIAGNOSIS AND TREATMENT OF NEOPLASMS OF THE CHEST WALL

With the exception of neoplasms of lymphatic origin and Ewing's sarcoma, thoracic wall tumors should be treated by complete excision.

Superficial Benign Tumors. It is important to emphasize that regardless of apparent superficiality, extirpation of a large sized thoracic wall neoplasm should not be regarded as a minor procedure. Sebaceous cysts and small superficial lipomas are usually easily removed and perhaps the caution of having available major operating room facilities is superfluous in these cases. It must be remembered, however, that even the innocent appearing lipoma may have an iceberg configuration into the pleura and that the extension into and through the chest wall may convert the so-called minor procedure under local

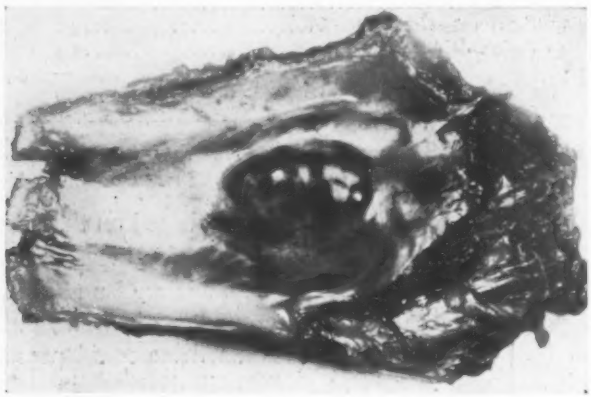


FIG. 1.—The appearance of a fibrosarcoma of the chest wall after block resection.

anesthesia into a painful, dangerous and inadequate operation. The cases of lipoma included in Table I are representative of lipomas which present major surgical problems. The following case is typical.

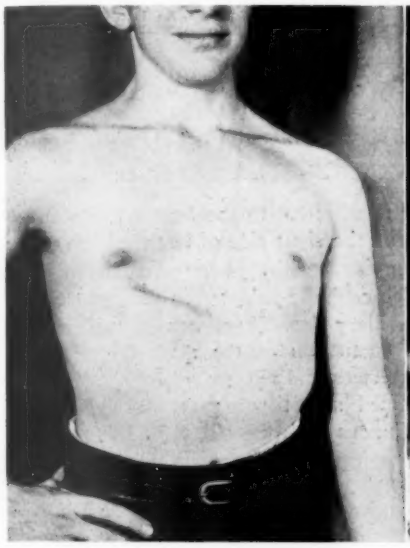
Case 1.—A 28-year-old colored female first noticed painless swelling in the left anterior chest wall seven months before admission to the hospital. The tumor grew rapidly and on admission the superficial portion of the lesion measured approximately 8 by 5 by 4 cm. The mass was situated just above the left breast and extended beyond the level of the clavicle. It seemed to be attached to the deeper structures of the supraclavicular space. The remainder of the history, physical examination and laboratory findings were not significant.

At operation the tumor was found to extend over the anterior chest wall into the supraclavicular space, where it extended over the clavicle deep in the neck to the level of the pleura at the apex of the thorax (Figs. 1 and 2).

After the tumor was removed its dimensions were found to be 16 by 12 by 5 cm. The lipoma was encapsulated by thin, transparent membrane. Its cut surface revealed yellow, lobulated fat and microscopic sections demonstrated adipose tissue.

The hazards of attempting to excise this benign lipoma without adequate facilities are obvious.

Other Benign Tumors. If lipomas are excluded entirely, nine, or approximately 45 per cent of the remaining 22 benign tumors listed in Table I are primary nerve tumors. These neoplasms are common and potentially dangerous lesions. Histological classification is difficult and the transition to malignant degeneration insidious. Recurrence of these lesions is inevitable unless complete excision is accomplished. There is no possible way to be certain of the exact nature of these tumors until tissue for microscopic examination is available. Proper treatment consists of complete resection of the lesion. These tumors may remain dormant for months or years and suddenly undergo rapid malignant degeneration.



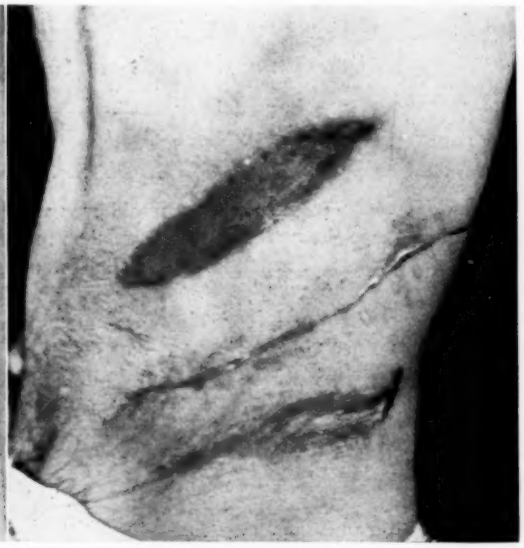


FIG. 2 FIG. 3

FIG. 2.—Removal of bony sections of three ribs, about half of the sternum and attached soft tissue was necessary to extirpate an anterior chest wall tumor. Note lack of deformity.

FIG. 3.—Photograph after healing of chest wall wound, which required releasing incisions and skin grafts.

The nature of osteochondromas and chondromas may be suspected from clinical findings and roentgen studies. Danger of malignant change and the high recurrence rate when inadequate excision is performed are comparable to those experienced with the primary nerve tumors.

Fibrous dysplasia of the ribs requires complete excision of the lesion. The diagnosis can usually be made by roentgen examination. It is interesting that in all of the cases of fibrous dysplasia of the ribs there was a history of severe trauma to the region involved.

The potential dangers of chest wall tumors which may be considered benign are illustrated by the following tragic case.

Case 2.—A young physician, age 25, injured his chest while skiing. A roentgen examination of the thorax following the accident revealed a tumor of the chest wall at the posterolateral aspect of the left fourth rib. A presumptive diagnosis of osteochondroma of the left fourth rib was made and the young man was advised by an eminent physician to disregard the lesion. Ten years later the patient complained of easy fatigability and low grade, unexplained fever. A complete examination including extensive laboratory tests failed to reveal the cause of the fever. Finally chest wall pain became evident.

The neoplasm was extirpated by block excision of the chest wall. The tumor was adherent to and appeared to have invaded the upper lobe of the lung and accordingly part of the lobe was excised. Microscopic examination of the tissue established the diagnosis of fibrosarcoma. The patient died approximately a year later of wide-spread metastases and local recurrence.

It is impossible to know whether the original lesion in this case would have been classified as a fibroma, neurofibroma or osteochondroma. This is of academic interest since the neoplasm degenerated into a highly undifferentiated sarcoma and killed the patient.

A second case of fibrosarcoma of the chest wall is presented to illustrate the possibilities of early, radical surgical intervention.

Case 3.—The patient, a 19-year-old woman, was admitted to the hospital less than three weeks after she noticed a small, painless mass on the right side of her chest directly beneath her breast. Examination revealed an otherwise healthy young woman with a tumor mass approximately 3½ by 2½ by 2 cm., which could be palpated in the right chest wall at the level of the seventh rib in the anterior axillary line. There was no tenderness, the skin was normal, there was a suggestion of fluctuation of the mass on physical examination. The lesion was attached to underlying tissues and rib.

Roentgen examination revealed a sharply defined soft tissue mass overlying the anterior end of the rib with dense calcification continuous with the rib and at the center of the mass. The rib appeared to be involved, showing indefinite markings and bony destruction (Fig. 3). The tumor projected into the chest.

Two days after admission a radical resection of the tumor was performed with wide excision of skin, underlying muscle, the sixth, seventh and eighth ribs and pleura in a block (Fig. 4). The postoperative course was uneventful. The patient left the hospital in ten days. Microscopic examination revealed a fibrosarcoma, perhaps originating in the periosteum of the rib. This patient is alive and well more than 5 years after operation.

Malignant Tumors. In more than 50 per cent of cases of malignant chest wall tumors, bone changes can be demonstrated on roentgen examination. Vague pain which becomes more severe and localized as the lesion progresses is common. Pleural effusion, severe pain and fever indicate inoperability and impending death.

It is important to record that in this group of cases of malignant neoplasms of the chest wall an average time of six months elapsed before the complaint of pain was investigated by roentgen examination. Approximately 75 per cent of these patients were treated for neuritis by various and devious methods. Neuritis therapy occupied an average of two months.

Only two of the patients included in Table II are alive. The complaint of discomfort in the chest wall in one instance prompted immediate roentgen examination which revealed bone destruction suggesting an osteogenic sarcoma. The other living patient is described in Case III.

The principles established by experiences with malignant or potentially malignant tumors elsewhere in the body are applicable to thoracic cage neoplasms. Repeated ineffectual blows may result in temporary palliative decisions but an immediate initial knock-out punch offers the only satisfactory solution. False fears of the consequences of resection of portions of the entire chest wall accounts for many incomplete operations. Case IV is an example of underestimation of the seriousness of chest wall tumors and the consequences of inadequate initial treatment.

Case 4.—A 32-year-old, white male noticed a painless swelling at the level of the tip of the left scapula. Vague pain of several weeks duration focused his attention on the region before the mass was evident. An original clinical diagnosis of lipoma was made and excision undertaken with local infiltration analgesia.

A cystic tumor extending beneath the scapula was encountered. The attempted dissection resulted in rupture of the mass, which contained dark bloody fluid. The tissue was excised and the wound closed.

Microscopic examination of the tissue established the diagnosis of malignant hemangioendothelioma.

Eight weeks later when the patient was first seen by us the only evidence of a chest wall tumor was a well healed scar about 10 cm. in length near the tip of the scapula. There was no palpable tumor and no hint concerning the possible boundaries of the original lesion.

Since the tissue excised at the first operation was malignant and available evidence suggested inadequate extirpation, a more extensive operation was recommended to the patient.

Reluctant co-operation was obtained from this apparently healthy young man and a block excision of the chest wall performed in the region of the original lesion. Absence of palpable or visible landmarks made it necessary to establish arbitrary limitations of dissection. The scar resulting from the original operation was considered the tumor site and the chest wall removed in a block with a margin of approximately 7 cm. around the scar. This included skin, scar, muscle, portions of three ribs and pleura.

Microscopic sections from the periphery of the specimen revealed many malignant cells. The patient died a few months later of generalized malignant disease. Roentgen therapy was not effectual.

Consolation may be found in postulating that the extremely malignant nature of this lesion precluded success no matter how early radical surgical therapy was undertaken. Facts, however, establish the serious error of operating upon any tumor without facilities for immediate radical resection of the lesion if it becomes necessary.

DIFFERENTIATION OF SURGICAL AND NONSURGICAL THORACIC WALL TUMORS

Unless the general physical condition of the patient precludes surgical intervention, all chest wall tumors require excision. If the lesion is considered malignant, concessions in operative risk are justifiable. The only known exceptions to this policy are primary Ewing's tumors of the chest wall and tumors of lymphatic origin. These should be treated by radiation therapy.

In this connection it is worth mentioning that attempts to perform radical operations for Ewing's tumor of the chest wall have been made. Our experiences indicate clearly that even in cases in which it appears that a Ewing's

tumor has been completely extirpated with a wide margin of normal tissue, the results are poor and no better than with roentgen therapy alone. Friedman and Blades[6] have combined radical surgical excision of Ewing's tumors with million volt roentgen therapy and again the results were unsatisfactory. It is possible that removal of a part of the chest wall in Hodgkin's disease might be desirable, but usually roentgen therapy alone is employed.

The importance of preoperative differentiation of tumors best treated by surgery from those which should receive roentgen therapy is apparent. Often clinical impressions are misleading, but in a high percentage of cases the roentgen diagnosis will furnish reliable clues. If Ewing's tumor or primary lymphoma is suspected but not proved, it is our opinion that a biopsy of the lesion should be obtained. Tissue obtained for rapid histologic sections may be difficult to classify as to the exact nature of the malignant cells, but Ewing's tumor and lymphomas can usually be identified. If this proves to be the case, the operation is terminated and roentgen therapy instituted. The presence of a neoplasm which does not respond to roentgen ray demands immediate radical resection of the tumor and surrounding chest wall.

Malignant Tumors. In two of the five metastatic tumors studied in this series, the true nature of the lesions was not discovered until tissue was available for microscopic section. In three instances metastases were known or strongly suspected. The case of metastasis from the thyroid is particularly interesting.

Case 5.—A thyroidectomy had been performed 8 years before the lesion involving the second rib was discovered. The roentgen diagnosis of the chest wall tumor was osteogenic sarcoma (Fig. 6). Accordingly, a block resection of the tumor involving removal of 3 ribs, pleura and chest wall was done. After the diagnosis was apparent, the records of the hospital in which the thyroidectomy had been performed revealed that a roentgen ray film of the chest had been taken which demonstrated a tumor of the rib. Apparently no one looked at the roentgenogram, or if this was done the presence of the rib lesion was ignored. The patient is well more than two years after operation and a complete survey has not revealed other metastasis.

Metastatic lesions from the thyroid may produce the roentgen appearance of osteogenic sarcoma. Ackerman[7] and others have described the osteolytic effects of these lesions. Primary disease in the thyroid gland should be considered in bone tumors with these characteristics.

The wisdom of attempting surgical extirpation of a single demonstrable metastatic tumor of the chest wall is debatable. With the exception of the case just described our results have been poor and it is questionable whether life has been prolonged by palliative operations. It is apparent, however, that if the risk of the operation is not excessive some benefits may be gained and little can be lost.

SURGICAL TECHNIC

Large segments of the thoracic cage can be resected with practically no functional disturbance and little deformity (Fig. 7). It is amazing, therefore, to find so many examples of inadequate "shelling out" procedures employed

in the treatment of deep chest wall tumors. Sometimes it is necessary to excise in a block long segments of several ribs with pleura, muscle and attached skin to obtain safe clearance of the lesion. In the extreme cases closure of the defect may present difficulties. Long releasing incisions through the skin and subcutaneous tissue above and below the original wound will allow closure, and skin grafts can be applied immediately to the defects (Fig. 8).

The same principles which were established in the management of large chest wall defects from war wounds can be applied in these cases. Almost any wound can be treated satisfactorily, regardless of its size, by plastic procedures involving only structures of the chest wall. The use of tantalum plates and other foreign bodies is unnecessary and unsatisfactory. Weak and soft defects can be minimized by shifting strips of periosteum from adjacent ribs and anchoring them in the wound. Transplants of fascia lata have been employed but are rarely necessary.

If there is intrathoracic projection of the tumor, the pleura should be entered at least two interspaces above or below the lesion. This will allow inspection and palpation of the deep portions of the neoplasm and establish boundaries for the block dissection.

If a biopsy has been necessary, the precaution of drape and instrument change should be observed and a wide margin of skin excised around the biopsy site if an operation is to be performed.

SUMMARY

Forty-seven per cent of the tumors described in this report were malignant. This figure has no statistical value. Exclusion of superficial lipomas, sebaceous cysts and the selection of deep or complicated neoplasms insures against exaggeration of the seriousness of chest wall tumors. Reasonably accurate preoperative segregation of superficial, benign lesions is possible, however, leaving the types of tumors which have been described. In this latter group malignant change is frequent, and radical aggressive therapy is indicated.

The structure of the chest wall allows complete anatomical extirpation of many tumors with sufficient adjacent healthy tissue. Apprehensions concerning deformity and disturbances in cardiorespiratory functions resulting from radical resections of the chest wall are not justifiable. It is possible that the common disappointing results following treatment of these lesions would be appreciably improved if more complete and radical operations were performed.

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DISCUSSION —DR. OSLER ABBOTT, Atlanta, Ga.: I should like to express my appreciation of the privilege of being allowed to speak as a guest. I hesitate to disagree with Doctor Blades. There are a few things I would like to mention that I think of importance in regard to this subject.

First, I think there is beginning to be a valuable place for the use of the laminagram in the study of lesions of the chest wall, particularly when one is confronted with the question of whether it is a deformity of the costochondral junction or an actual tumor. This is still in process of evaluation, but it is particularly helpful in picking up areas of calcification which could not be seen otherwise, and is similar to work which we have been doing recently in diagnosing dissecting aneurysms by the use of the laminagram. The second thing I would like to mention is in rather complete disagreement with Doctor Blades on the combination of radiation therapy and excision. Particularly in malignant lesions of the chest wall, liable to marked increased vascularity, if we get into those without a protective surrounding sclerosis the chances of disseminated spread through the blood stream are so great that I think it is fallacious to treat them in any manner other than very intensive radiation therapy beforehand, then a wide block excision. We feel we have had some success with Ewing's tumor by that means. We recommend using about twice the ordinary dosage of radiation therapy as a preoperative measure.

The final thing I want to mention is that, particularly in the Veterans Facility, we are constantly confronted with a painful chest wall in the presence of large and major defects. A method of palliation must be achieved, for these complaints are not purely on a compensation neurosis basis. We are still trying to find a method, but are beginning to feel that tantalum mesh is the best method, and frequently it need not be removed if satisfactorily implanted. Another trick of tremendous help in the lower ribs, when one is trying to reconstruct the chest wall, is complete excision of the myochondrosis. Even after three ribs have been removed the chest wall can be drawn together in a firm and satisfactory manner.

DR. JOHN S. PAUL, Washington (closing): In treating these chest wall tumors we have attempted, so far as possible, to stay away from the biopsy as ordinarily considered. We have preferred, in these patients with chest wall tumors, to prepare them initially for radical resection and then, at the time of operation, do a wide excision type removal rather than biopsy. We have reserved the actual biopsy, as ordinarily considered, for those tumors which the radiologist has informed us preoperatively are probably Ewing's sarcoma. In those, biopsy is done at the time of operation; if the frozen section report comes back as Ewing's sarcoma, we have simply closed the chest and subjected the patient to radiation.

CONSERVATIVE RESECTION OF CHRONIC LUNG ABSCESS*

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SURGICAL TREATMENT offers the best chance for survival and cure of pulmonary abscesses. In clinics where this fact is appreciated, a marked reduction in mortality, and a substantial increase in cure rate of this dread disease, has been realized through open drainage of acute abscesses and resection of chronic abscesses. Other plans of management viewed through large series of collected cases have resulted in a high mortality throughout the past two decades.[2, 17]

Acute lung abscess has been treated with best results by early diagnosis, prompt intensive therapy, accurate localization and open drainage during the first six weeks from the onset of symptoms.[3, 10, 12, 13, 15] This improvement in the treatment of acute lung abscess with a mortality in the neighborhood of 5 per cent is gratifying. Indeed, with the correspondingly high cure rate reported, it is possible that the problem of chronic lung abscess may be virtually eliminated. Unfortunately, this is not to be expected until the results of proper treatment are more widely acknowledged.

Chronic lung abscess presents a more complicated problem. In the acute type the cavity is usually unilocular. Daughter abscesses appear in the chronic phase from bronchial obstruction and direct spread across incomplete fissures. The latter pathway is especially important in the right lung, where a high incidence of incomplete fissures makes it possible for all three lobes to be involved by direct extension of an inflammatory process.[4] Fibrosis develops in the abscess wall and surrounding lung parenchyma; atelectasis and neighboring bronchiectasis appear. In addition to these local changes, the systemic effects of chronic sepsis occur.

The structural changes in the lung parenchyma are marked and so well established that healing will not result usually when a simple drainage procedure is employed. This form of treatment in the chronic phase is associated with a mortality rate of about 30 per cent, with cures in from 25 to 35 per cent of the survivors.[14] On the basis of these discouraging results and the marked structural changes within the lung, various forms of resection have been utilized with better success. Favorable results have been reported by Shaw and Paulson,[16] Lindskog,[9] Kent and Ashburn,[7] Neerken and Grow[11] and others,[1, 8] by removal of the involved pulmonary segment, lobe or whole lung, with mortalities ranging from 3.8 to 7.9 per cent and a correspondingly high cure rate. Glover and Clagett[6] similarly have reported 37 cases of chronic lung abscess treated by resection, and in the 21 requiring removal of a lobe

* Read before the Southern Surgical Association, Hot Springs, Virginia, December 8, 1949.

or a part of a lobe a mortality of 3.8 per cent resulted. In those cases requiring bi-lobectomy or pneumonectomy the mortality was about ten times as great.

In the more complicated abscesses involving two or more lobes, extirpation requires extensive surgery in a poor risk patient and frequently the sacrifice of much functional pulmonary tissue. Attempts to confine the removal to diseased tissue alone by multiple segmental resections are often prolonged, shocking procedures, by virtue of the altered anatomy and increased friability occasioned by the inflammatory process. The simple excision of a complicated abscess in such a situation seems to be a possible solution. A procedure of this sort should have the advantages of preservation of pulmonary parenchyma and of less surgical trauma. The extirpation of abscess cavities has been utilized in other organs of the body, notably the brain, in which the demand for

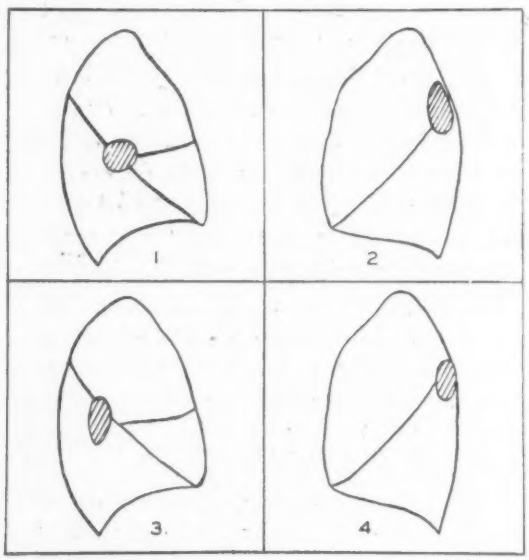

FIG. 1.—Diagram showing the location of the abscesses in the case reports.

preservation of functional tissue has no peer.[5] The idea of applying this form of conservative surgery in the lung originated from the unusual success with which it has been employed in the central nervous system.

Briefly the excision as utilized in the four cases reported here is accomplished in the following manner: Thoracotomy under endotracheal anesthesia is carried out through a resected rib bed from the posterolateral approach. The limits of the abscess are outlined by palpation. Irrespective of its extension into multiple lobes, the confluent mass is extirpated by division of lung tissue in the zone just adjacent to the abscess between suture ligatures of fine Deknatel. Gentle positive pressure is applied through the endotracheal system after excision to ascertain the proper inflation of the remaining pulmonary

tissue. The wound is closed in layers, and suction catheter drainage is maintained for 48 to 72 hours postoperatively.

Certain theoretical hazards in such a technic are obvious. The dissection is guided not by a true anatomical approach but rather by the limits of an inflammatory reaction, introducing the risk of compromising distal pulmonary tissue by interruption of the vascular or bronchial supply. Actually, the procedure is a modification of a segmental resection by virtue of the fact that abscess cavities begin in and are roughly defined by pulmonary segments.

FIG. 2.—Illustrative photograph showing the technic used in removing portions of a lobe.

The dissection in inflammatory tissue also appears hazardous because of gross contamination and the destruction of natural barriers to the spread of infection. Thus far, no case has developed distal atelectasis or gangrene, nor has empyema been a complication. The rough segmental limits of the dissection, chronicity of the infection and liberal use of chemotherapeutic and antibiotic agents have apparently obviated these hazards. Glover and Clagett[6] have reported two similarly treated cases without mortality or morbidity and a 100 per cent cure rate. Overholt and Rumel[13] reported two cases in which a portion of a lobe was thus excised with one cure and one death from a later secondary brain abscess and septic meningitis.

Four patients with chronic complicated lung abscess have been subjected to this form of resection. The postoperative mortality has been zero, morbidity rate zero, and although the period of follow-up has been short, the apparent cure rate is 100 per cent. The selection of these cases requires that preoperatively no widespread associated bronchiectasis or multiple distant abscesses can be demonstrated. At operation, selection is made in the presence of an abscess involving portions of two or more lobes, or a limited and accessible area of one lobe, and in which no obvious large bronchovascular structure lies in the line of excision. In the case of single lobe abscesses, except for minimal involvement, lobectomy is probably the operation of choice.

The following case reports are submitted as illustrative of this form of excision of chronic lung abscesses.

Case 1.—M. N. (Guilford County Sanatorium.) A 31-year-old male coal miner was admitted complaining of a productive cough and intermittent hemoptysis of 8 months duration and a progressive weight loss of 13 pounds. Five months prior to admission a chest roentgenogram showed a cavity in the right lung field. Physical examination revealed dirty, carious teeth, marked clubbing of the toes and fingers, and bronchial breathing in the right posterior mid-lung field. A tuberculin test was negative. Roentgen ray films of the chest showed a large cavity in the right lung field, which appeared to be in the apex of the right lower lobe. Bronchoscopy showed much purulent secretion from the upper lobe bronchus and the superior bronchus of the lower lobe on the right side; bronchograms were negative.

A right thoracotomy was done through the posterolateral seventh rib bed; no adhesions to the parietal pleura could be found, but an indurated area 5 cm. in diameter was encountered at the junction of the short and long fissures, obliterating these fissures and extending into the three lobes for an equal distance. By means of suture ligatures placed in the zone just proximal to the indurated area, the mass was isolated and excised. All lobes were inflated properly at the end of the procedure, and layer closure of the wound with suction catheter drainage was instituted. Convalescence was satisfactory and he was discharged on the sixteenth postoperative day afebrile and without sputum. A roentgenogram of the chest on the fourteenth postoperative day showed complete re-expansion of the right lung, with haziness throughout the lung field from lipiodol. The patient has subsequently returned to work as a coal miner and has remained well for 10 months.

The pathology report confirmed the diagnosis of chronic pulmonary abscess with organization of the wall, chronic pneumonitis with fibrosis, and anthracosis.

Case 2.—R. S. W. (N. C. B. H. No. 75939), a 22-year-old white female, was admitted to the N. C. Baptist Hospital complaining of a productive cough of 8 months' duration, one episode of hemoptysis 3 months prior to admission, and a 22-pound weight loss. Bronchograms and sputum studies done in another hospital were suggestive of chronic lung abscess, and she was referred for treatment. Physical examination was not remarkable, nor were the routine laboratory studies. Repeated sputum examinations were negative for acid fast organisms, and the tuberculin test was negative. A roentgenogram of the chest disclosed what appeared to be a chronic lung abscess in the right mid-lung field. Bronchoscopy showed pus in the right upper and right lower lobes, and bronchograms were negative.

Right thoracotomy through the sixth rib bed from the posterolateral approach was performed. There were dense adhesions to the parietal pleura along the interlobar fissure between the upper and lower lobes just posterior to the short fissure. These adhesions were severed and revealed the interlobar fissure in this location to be obliterated. There

FIG. 3

A B

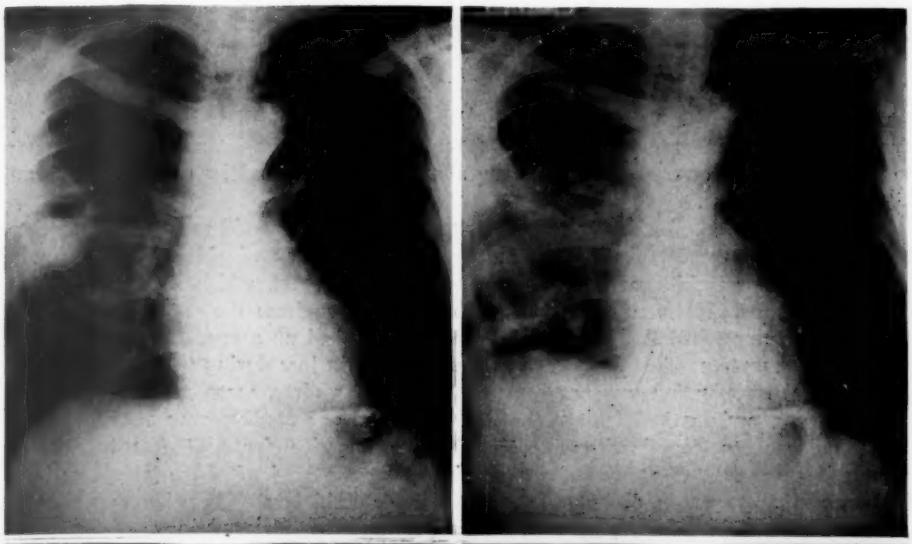

FIG. 4

A B

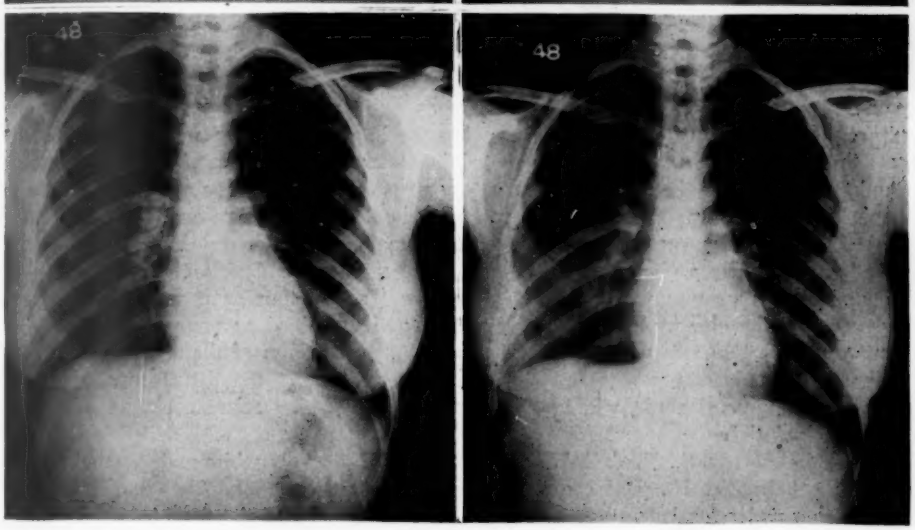

FIG. 3.—(Case 1) (A) Preoperative film showing the abscess cavity. (B) Postoperative film.

FIG. 4.—(Case 2). (A) Preoperative film. (B) Follow-up film two months after excision of the abscess.

was induration in the right lower lobe with extension of the induration into the right upper lobe, involving an area roughly 8 cm. in diameter. An excision of the indurated area was elected and carried out in the usual fashion. The larger portion of the abscess in the lower lobe was removed completely by excision, and a portion of the abscess wall in the upper lobe which remained was cauterized with phenol and alcohol. The remaining lung tissue re-expanded completely, closure was accomplished, and tube suction drainage instituted.

Convalescence was without event. There was no fever or sputum on discharge on the tenth postoperative day. The patient has remained well during 21 months since operation. Roentgenograms of the chest taken 2 and 7 months later showed the lungs to be clear. The diagnosis of chronic lung abscess was confirmed by microscopic examination of the tissue.

Case 3.—(H. B. B., N. C. B. H. No. 96845). A 58-year-old male textile worker was admitted to the N. C. Baptist Hospital with a history of six weeks previously having experienced chills and pleuritic pain in his left chest, which subsided on conservative therapy. His cough had been mildly productive from the onset of symptoms until 8 days prior to admission, when he suddenly coughed up a cupful of thick, blood-streaked, foul sputum. There had been a 12 pound weight loss during the present illness. The physical examination was not remarkable, save for dirty, worn and carious teeth, infected gums, and marked fetor oris. Routine laboratory studies were within normal limits and sputum examinations were repeatedly negative for acid fast bacilli. A roentgenogram of the chest showed a cavity in the medial one-half of the left upper lung field measuring 2 cm. in diameter and containing a fluid level. Bronchoscopy showed pus and blood in the left main stem bronchus with no other abnormalities.

Left thoracotomy through the posterolateral sixth rib bed was performed. Dense, firm adhesions were noted between the posterior surface of the upper and lower lobes and the parietal pleura. These were severed by sharp dissection and a small amount of purulent material was encountered which was subsequently found to be sterile on culture. A 10 cm. area of induration crossing the interlobar fissure was found beneath the adherent area, about half being situated in either lobe. Segmental resections were abandoned because of the marked inflammatory reaction and distortion of the hilar structures. The abscess was excised in the zone proximal to the induration by incision and suture ligation in sequence. Re-inflation of the remaining pulmonary tissue was accomplished satisfactorily and the wound closed in layers around double thoracotomy tube drainage. Recovery was without event. There was no sputum or fever when he was discharged on the eighth postoperative day. The patient gained 22 pounds in the subsequent 2 months. He was symptom free and a roentgen ray film of the chest showed the left lung field to be practically clear. He has remained well 6 months postoperative.

The diagnosis of chronic lung abscess was confirmed by microscopic examination of the excised tissue.

Case 4.—(M. Q. S., N. C. B. H. No. 98966). A 68-year-old white male was admitted to the N. C. Baptist Hospital complaining of a productive cough. Two months prior to admission under local anesthesia, he had had an abscessed tooth extracted which was complicated by swelling and pain in the jaw with fever, for which he was given penicillin with gradual resolution. Two weeks later he noted a cough productive of foul, blood-streaked sputum. At this same time, he experienced pain in the left side of the chest, began to run a low grade fever and lost about 15 pounds in weight during the following 6 weeks. The physical examination was not remarkable except for an emphysematous thorax and dullness to percussion with diminished breath sounds over the left lung base posteriorly. A roentgenogram of the chest showed a pleural effusion on the left side which yielded 500 cc. of a sterile, cloudy yellow fluid on aspiration. A follow-up film disclosed multiple fluid levels in the left lower lung, probably in the superior segment of the lower lobe. He was treated with postural drainage, penicillin, sulfadiazine, and aureomycin without demonstrable benefit.

FIG. 5

A B

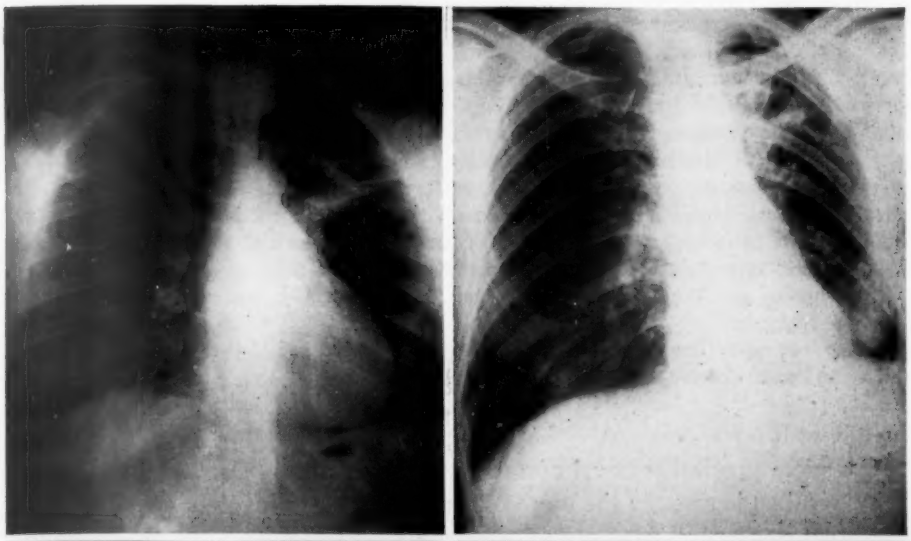

FIG. 6

A B

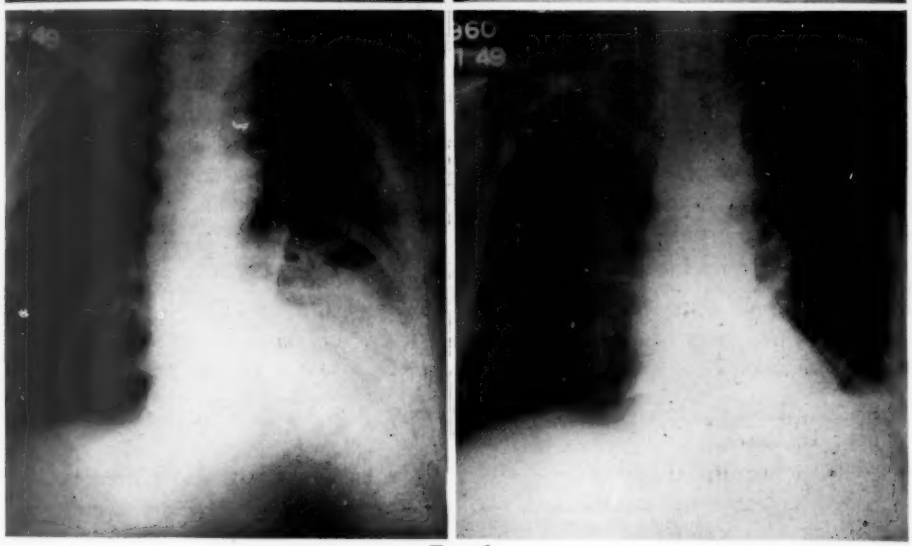

FIG. 5.—(Case 3). (A) Preoperative lordotic film. (B) Conventional chest film one month after excision of the abscess.

FIG. 6.—(Case 4) (A) Preoperative film showing pleural effusion and multiloculated appearance of left lower lung field. (B) Chest film two weeks after excision of small abscess in dorsal apical segment of left lower lobe and decortication of left lower lobe.

Routine laboratory studies were within normal limits except for a polymorphonuclear leukocytosis. Sputum studies were negative for acid fast bacilli. The fluid aspirated from the chest was found to be sterile and negative for acid fast organisms on smear. On bronchoscopy, moderate amounts of mucopurulent secretions were present in the left main and lower lobe bronchi.

Left thoracotomy through the bed of the eighth rib was performed and 800 cc. of an odorless cloudy fluid was encountered, which subsequently proved to be sterile. There was a fibrinous exudate over the pleural surface of the left lower lobe which was removed without difficulty. In the dorsal apical segment of the left lower lobe a cavity with minimal surrounding induration was palpated and was excised. The remaining lung tissue re-expanded satisfactorily, layer closure of the wound was accomplished, and tube suction drainage instituted. The patient's postoperative course was without event and he was discharged without fever or sputum on the eleventh postoperative day. The left lung remained expanded, and aside from an area of hypo-aeration and thickened pleura along the lateral margins of the left lower lobe the chest roentgenogram was negative at the time of discharge. In the intervening 5 months he has remained well and symptom free.

Examination of the excised tissue microscopically showed slight interstitial fibrosis and a moderate chronic inflammatory reaction.

SUMMARY

Complete surgical removal of a chronic lung abscess offers the best chance for survival and cure. In the more extensive abscesses involving two or more lobes, removal by bi-lobectomy or pneumonectomy carries a greater mortality because of the magnitude of the procedure in a debilitated patient. The sacrifice of functional pulmonary tissue in such instances augments the surgical insult. Attempts at conservatism through single or multiple segmental resections are often disappointing due to the difficulty of accomplishing an anatomical dissection in the presence of marked distortion and free bleeding attendant to an inflammatory process. Simple excision in selected cases warrants consideration to obviate these hazards associated with the removal of chronic lung abscesses. Complete removal is easily accomplished, the operative insult is reduced, and functional lung tissue preserved by such a procedure.

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DISCUSSION.—DR. J. S. HARTER, Louisville, Ky.: We have used this method of taking out lung abscesses on a few occasions; one recently in which the abscess contained about 700 cc. of pus. In an abscess that is not draining, of this type, the amount of pus in the abscess or the size of the abscess does not represent that much lung destruction. This was excised from the upper lobe and the lower lobe on the right, posterior to the middle lobe. The patient made an uneventful recovery and at present shows only a little scar tissue in that region. We have carried that out in several instances with small abscesses, certainly. Ordinarily, however, as Doctor Bradshaw will agree, I am sure, chronic lung abscesses are so frequently accompanied by bronchiectasis and fibrous destruction of the lobe that it is better to do a lobectomy than an excision. But there are certainly some instances in which this operation is applicable.

DR. J. E. DAILEY, Houston, Texas: I was much interested in Doctor Bradshaw's presentation. I would like to have heard the paper in toto because a few questions occurred to me. We have had some experience with local excision of abscesses such as he described and almost always were unfortunate enough to have complications such as persistent bronchopleural fistulas, and some that required thoracotomies and long drainage afterwards. I would like to ask Doctor Bradshaw if he could, in summarizing, give us some idea of the percentage of such complications occurring with the technic of local excision of these abscesses.

Lately, in most instances, I have found that we could do a segmental resection or, if that were too difficult, I have not hesitated in taking out the entire lobe. I do not feel quite the concern about loss of pulmonary tissue that some of the men do. It is certainly technically easier to take out a lobe than it is to do a segmental resection or even a local excision of a portion of lung.

DR. H. H. BRADSHAW, Winston-Salem (closing): I want to thank Doctor Harter and Doctor Dailey for their remarks. Certainly if the disease is limited entirely to one lobe, one would not think of this type of excision very often. It is the abscess that crosses the fissures in a person who would be considerably crippled by having two or more lobes removed, in which conservative resection is especially useful. As to infection after this has been done, so far this has not been a problem. The pleural cavity has been drained for about 72 hours after operation. Needle aspiration is occasionally needed thereafter; but frank empyema has not yet been a problem. It seems likely with more experience that empyema will occasionally be present.

The point Doctor Harter brought up, about there being bronchiectasis with these abscesses, must be kept in mind. The procedure is valuable only with localized processes, and not in the presence of widespread bronchiectasis or smaller abscesses in other lobes. This procedure, I think, has a fairly limited use, but I believe it does have that use.

Editorial . . .

The Surgical Treatment of Cholelithiasis

The surgery of cholelithiasis has two objects. It is concerned primarily with the health or the rescue of the individual patient, and, secondly, as in all surgical procedures, research, no less than therapy, should be the aim.

In the days to which we look forward with hope, and no little expectation, it is possible that the recognition of earlier stages in this disease may enable us to prevent, or to delay, those advanced or terminal complications with which our therapeutic activities are now engaged, i. e., to recognize precalculus cholecystitis.

By the time choleliths have developed, medicinal therapy has nothing more to offer than to insure a degree of clinical silence; it is not able to retard the steadily progressive pathologic changes. It is a well recognized fact that the symptoms of this disease syndrome may be in abeyance, or more rarely, their complete relief may be noted while the morbid processes continue steadily to develop in the walls of the gallbladder and the ducts until finally cancer reveals itself. The conditions which make it necessary to decide against surgical relief must be extremely few. The danger in the operative treatment of cholelithiasis comes from delay, which results in infection of the ducts, damage to the liver, deposition of stones in the intra- and extrahepatic ducts and, finally, the possible development of the cholelithic heart. Yet the dangers of operative treatment *should* be small indeed, and operation is not only safer than continued medical treatment, but it is far more merciful. On the other hand, the surgery of cholelithiasis is difficult, sometimes extremely difficult, and is thought by those most experienced in this field to be of greater technical difficulty and to present more problems for immediate accuracy of judgment than any other branch of surgery. The operator must be constantly on the alert, for contained within an operative field of relatively small dimensions there are structures, damage to which may be irremedial and irreparable. Operations upon the biliary tract should not be attempted by surgeons unless they have had great and continued opportunities for operative work in this field. Unfortunately, as attested to by reports from various clinics in the United States, patients suffering from cholelithiasis have been operated upon unsuccessfully, either as a result of inadequate or inaccurate surgery, by inexpert or untrained operators who are incompetent when confronted with conditions which may tax all of the energies and call out all of the reserves of even the most efficient and well-trained surgeon. Surgery is not only a craft—although craftsmanship of a high order is essential—it is also an art, and one must distinguish between the artist and the artisan. Men, after completing a rigid and long training under close supervision by a master surgeon, should feel towards surgery a complete devotion as well as a sense of special dedication. The practice of surgery should demand the most flawless integrity in thought and act. No

one acquainted with the trust can deny that far too many operations have been performed by those who, having perhaps had a measure of success in trivial or small cases, rashly embark upon procedures which they are not fully competent to undertake. The surgeon who pretends to assume the responsibility of operations upon the biliary tract, facing the decisions that must be made in a very short space of time and exercising what would be recognized as sound surgical judgment, must have an intimate knowledge not only of the normal anatomy of the biliary tract but also the physiology of the liver and the adjacent structures and organs.

In the last two decades great forward steps in surgical progress have been made because the introduction of better methods of sterilization and asepsis, the rapid development of anesthesiology, the blood bank, antibiotics and the sulfa drugs have greatly expanded the field of surgical endeavor. The dangers of operations on the heart, lungs, and intestines have been lessened, and the scope of the operative surgeon has been widened. These remarkable and brilliant advances in those fields of surgery have so completely occupied the attention of physicians and surgeons alike that the yet unsolved fundamental problems concerning operative procedures less spectacular are, so to speak, thrust in the background.

Even though there is abundant literature on the surgical treatment of gallstones, there remain many controversial points. To such procedures clings an aura of uncertainty and indecision. There is a lack of agreement among various surgeons, as well as divergence in the philosophic and therapeutic rationale of different clinics in the United States and abroad. In support of the criticism of the surgical treatment of cholelithiasis in the United States, one might refer to the great increase in reports of irreparable operative damage to the common and common hepatic ducts in the surgical literature. The many ingenious and mainly unsuccessful operations suggested to correct this catastrophic surgical blunder are mute testimony to the mistakes of tyros and of a group who might be classified as "merchant surgeons." Such cases as appear in the surgical literature, of course, do not include the silent mortality, which we may be sure exists. Unfortunately, poor results in surgery are rarely reported.

Personal conversations with prominent surgeons from Scandinavia, the British Empire and the Continent leave one with the impression that operative damage to the extrabiliary ducts is far less frequent in their countries than here. Their literature contains fewer references to the repair of the ducts damaged during removal of the gallbladder. In this respect one can only conclude that the training and supervision of some surgeons in the United States is far from adequate.

Another factor which undoubtedly plays no small role is the indifference on the part of the referring physician. This indifference springs from the fact that in the last two decades the surgical treatment of gallstones has been regarded as a commonplace operation that is relatively simple in its execution—that *almost any so-called surgeon can remove a gallbladder.* Many medical men whose attendance in the operating room is infrequent indeed freely advise

patients as to the operator they should employ, when their knowledge of surgical technic or the respective surgeon's ability is essentially non-existent. Thus, this most difficult surgical field, in which so many vital structures are found, whose damage can be fatal, immediately or remotely, and which is generally irreparable, is blundered into with complete abandon by some operators who have little or no experience. When injuries to the extrahepatic ducts reveal themselves by the presence of drainage of bile or the onset of obstructive jaundice in the immediate postoperative days, the patient is then beguiled, deceived and misled with statements and excuses that there was an abnormality of the ducts or that obliterative inflammatory processes had obstructed the common hepatic or common duct and that further reconstructive operations will have to be performed.

Regardless of the method employed, such reconstructive procedures are not only extremely difficult in even the most experienced hands but, unfortunately, with all such procedures there is often a high percentage of failures. If an unsatisfactory operation has been performed on the lungs, the stomach, or other parts of the gastro-intestinal tract, serious mistakes may often be corrected by employing the services of a surgeon properly trained in the affected field. However, since there exists only one common hepatic duct approximately one inch long and one common duct not more than three inches long, the magnitude of the surgical disaster and clinical calamity which may follow operative injury to these ducts is readily appreciated.

The employment of the various operative procedures described for the surgical treatment of cholelithiasis in the last 50 years offers the operating surgeon enough technical latitude to exercise good judgment and discretion, depending on the age, the clinical condition of the patient, and the pathologic findings. In acute inflammation of the gallbladder in older people it may often be wise to employ cholecystostomy in place of cholecystectomy, especially when the patient is critically ill. In the event of severe infection and gangrene, cholecystostomy may be preferred if changes in the gallbladder wall would render cholecystectomy an unwise procedure due to prolongation of the operative time and/or the spread of infection. Again, in the uninfected gallbladder in young people in their second and third decades, in which a solitary cholesterin stone might be present and in which the gallbladder itself has a normal blue color, it might be wise to perform a cholecystotomy for the removal of a stone. It is to be recalled that the gallbladder represents the only switch on a single-track road and in later years it might prove itself useful in case of the formation of neoplastic or some other types of common duct obstruction.

In the operation of cholecystectomy there are dangers to be avoided and there are many things to be remembered by the operating surgeon, who should be constantly alert during the dissection. In this operation more than in any other abdominal operation the exact position and relation of every structure encountered must be clearly defined. Not only the anomalies of the extrahepatic ducts such as the cystic, common hepatic, and common duct must always be held in mind, but also the possible presence of accessory extrahepatic ducts

which may occur in as many as 15 per cent of patients. These accessory ducts, it is well known, may vary from the size of a small linen thread to the size of a right or left common hepatic duct. In all probability, in a small percentage of the cases, the profuse drainage of bile which not infrequently follows cholecystectomy is due to the fact that one of these accessory ducts has been divided unknowingly by the operator. It is for this reason, of course, that the abdomen should always be drained by at least two, if not three, protective drains or cigarette localizers. The great majority of the accessory hepatic ducts come from the right lobe of the liver and more frequently than not are unrecognized at the time of removal of the gallbladder. The profuse drainage of bile following removal of the gallbladder is probably more often due to division of one of these ducts than to the loosening or pushing off of the ligature placed about the cystic duct, which has been advanced heretofore as the cause of unusual drainage following cholecystectomy.

On one point most surgeons with the greatest experience in dealing with the gallbladder and the extrahepatic ducts agree: *the abdomen should never be closed without drainage*, for it certainly does no harm to leave drains in for a few days, and it will prove to be a lifesaving measure if, unwittingly, accessory ducts have been divided. Furthermore, abnormalities of the right and left hepatic arteries, the cystic arteries, and even of the gastroduodenal and pancreaticoduodenal vessels must be held in mind. Not infrequently, the right hepatic artery, particularly in elder people, may become tortuous and have a caterpillar curve in it, that is, the curve like the arched back of a caterpillar in progression. This right hepatic artery may very well be mistaken for the cystic artery. Only careful dissection will reveal the small cystic artery emerging from the caterpillar-like arch. Ligation of the right hepatic artery may have severe consequences and presumably has been the cause of so-called postcholecystectomy liver deaths. The ligation of the right hepatic artery is now inexcusable. Division of this vessel in the patient whose liver cells are already damaged because of inflammation and stasis of bile may be a serious, or even fatal, matter.

Unanimity of opinion of even the most experienced operators, is lacking in regard to the necessity for the drainage of the common duct following choledochostomy, hepaticostomy, or drainage of the common hepatic duct. Even if drainage of the common duct is instituted there still seems to be a difference of opinion about the type of drain to be employed and the manner of its insertion into the duct to be drained. Only careful follow-up studies in the future will finally decide this long-continued controversial discussion of whether to drain or not to drain the extrahepatic bile ducts. The only necessity for drainage would seem to be the prevention of tension within the ducts, and it is questionable whether this tension may ever become so great as to be of any consequence, since the patulous ampulla of Vater, due to stretching of its fibers in the course of a thorough exploration of the common duct, will insure drainage of the bile into the duodenum, which is far more physiologic than to allow it to escape to the exterior. Another argument which has been

raised against the placing of tubes into the common duct is that they carry infection from the duct into the wound and vice versa. Although there have been many reports and discussions of the surgical procedures used in the treatment of cholelithiasis and many contributions by surgeons of great renown over the half-century, there still are controversial points and differences of opinion that remain to be settled.

Finally, it may be said that, while undoubtedly consistency has its virtues, in the surgical treatment of affections of the biliary tract one must have the ability to change one's mind and to learn by experience. For this there is no substitute.

WILLIAM F. RIENHOFF, JR.

EDITORIAL ADDRESS

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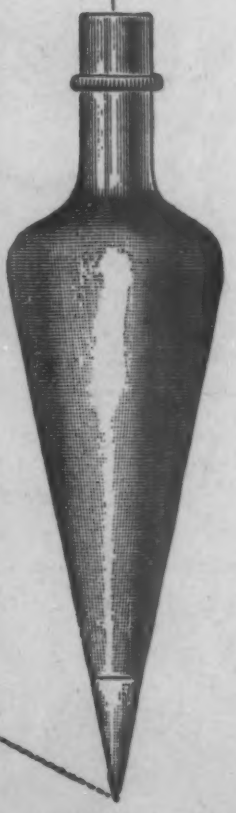

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